

S U P P L E M E N T to

PATHOLOGY and the CONSERVED OVARY

(A Statistical Survey)

from the thesis

OVARIAN ACTIVITY FOLLOWING HYSTERECTOMY

by

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In that part of the thesis entitled "Pathology and the "Conserved Ovary" an attempt has been made to assess the incidence of pathological change in ovaries conserved at hysterectomy. The investigation takes the form of a statistical survey based on the records of the Glasgow Royal Samaritan Hospital for Women, and in the following pages are listed the initials, ages and clinical numbers of every patient whose case records were scrutinised in order to obtain the necessary information.

This supplement, in fact, supplies details of the evidence on which the conclusions of the survey have been built.

It lists 1,215 patients who underwent the operation of hysterectomy with conservation of one or both ovaries, and indicates those who had to be readmitted on account of ovarian pathology.

It also enumerates, in chronological order, 872 patients who underwent the operations of oophorectomy, ovariectomy or simply laparotomy for what appeared to be ovarian pathology, and states in each case, whether or not the patient had previously undergone the operation of hysterectomy.

Cases of Subtotal Hysterectomy with Conservation  
of Both Ovaries

(Dr. McIntyre's Unit) 1927-48

1927

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Number in Hospital Records</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
1	Mrs. A.McL.	40	16	Fibroid	Nil
2	" E.McK.	42	23	Chronic subinvolu- tion	"
3	" J.R.	50	35	Do.	"
4	" M.P.	34	76	Do.	"
5	" M.T.	49	113	Fibroid	"
6	" M.B.	47	111	Do.	"
7	" M.M.	53	131	Chronic Metritis	"

1928

8	" M.S.	38	272	Chronic Metritis	"
9	" M.H.	28	276	Do.	"
10	" C.W.	36	281	Do.	"
11	" C.G.	52	325	Perforation of Uterus	"
12	" C.McG.	27	203	Chronic Metritis & Endometritis	"
13	" M.P.	26	407	Do.	"
14	" J.C.	47	470	Chronic subinvolu- tion	"
15	" M.M.	41	489	Fibroid	"
16	" A.A.	42	492	Chronic subinvolu- tion	"
17	" J.Mc.A.	23	359	Chronic Metritis	"
18	" A.D.	40	566	Fibroid	"
19	" J.M.	37	569	Chronic subinvolu- tion	"
20	Miss A.C.	42	576	Multiple Fibroids	"

1929

21	Mrs. L.D.	33	582	Cyctic glandular Hyperplasia	"
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22/



<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Number in Hospital Records</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
22	Mrs. M.T.	37	619	Chronic Metritis	Nil
23	" M.L.	35	779	Metritis	"
24	" A.C.	38	794	Fibroid	"
25	" R.M.	32	926	Fibroid	"
26	" A.M.	42	957	Adenomyoma	"
27	" M.D.	36	958	Chron. Endometritis	"
28	" U.S.	29	992	Endometrial Hyper- plasia	"

1930

29	Mrs. M.M.	37	1097	Chron. subinvolution	"
30	" A.O.	35	330	Do.	"
31	Miss C.D.	35	1126	Fibroid	"
32	Mrs. E.E.	38	1292	Chron. Metritis	"
33	Miss M.S.	35	1229	Chron. Endometritis	"
34	Mrs. E.W.	42	1328	Fibroids	"
35	" G.P.	27	1336	Subinvolution	"
36	Miss J.W.	28	134	Mult. Fibroids	"
37	Mrs. R.McA.	39	61	Chron. subinvolution	"
38	" E.M.	37	1482	Do.	"

1931

39	Mrs. N.R.	42	1576	Chron. subinvolution	"
40	" S.McK.	35	1669	" Metritis	"
41	" E.C.	39	1291	" subinvolution	"
42	" A.T.	33	1986	Mult. Fibroids	"
43	" E.J.	52	2015	Chron. Metritis	"

1932

44	" A.K.	44	2082	Chron. Metritis	"
45	" M.B.	42	2173	Chron. subinvolution	"
46	" M.O.	39	2248	Mult. Fibroids	"
47	" M.C.	38	2439	Do.	"

1933

48	" M.W.	36	2884	Chron. Metritis	"
49	" M.M.	40	3010	Do.	"
50	" V.B.	37	3013	Mult. Fibroids	"
51	" M.C.	32	205	Chron. Metritis	"
52/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Number in Hospital Records</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
52	Mrs. A.B.	33	3148	Mult. Fibroids	Nil
53	" C.L.	33	3186	Chron. subinvolution	"
54	" H.McC.	35	3328	" Metritis	"
55	" A.McG.	43	1490	" subinvolution	"
56	Miss E.K.	37	3410	Fibroids	"
57	Mrs. E.B.	41	3436	"	"
58	" I.H.	35	3462	Chron. subinvolution	"

1934

59	Mrs. M.D.	39	3154	Chron. subinvolution	"
60	" J.R.	37	3141	Fibroids	"
61	" S.C.	49	4341	Chron. subinvolution	"
62	" I.B.	41	4387	Do.	"
63	" M.C.	28	4386	Do.	"
64	Miss B.F.	29	4414	Fibroids	"
65	Mrs. J.T.	32	419	Chron. subinvolution	"
66	" G.H.	41	4426	Fibroids	"
67	" M.G.	34	4448	Do.	"
68	" A.G.	32	4486	Mult. Fibroids	"
69	" E.F.	42	4494	Chron. subinvolution	"
70	Miss M.C.	35	3341	Mult. Fibroids	"
71	Mrs. C.B.	34	4632	Chron. subinvolution	"
72	" C.McI.	27	4662	Cystic Glanduar Hyperplasia	"
73	" E.L.	40	4702	Mult. Fibroids	"
74	" A.G.	33	4799	Fibroids	"

1935

75	" H.McC.	35	5050	Cystic Glandular Hyperplasia	"
76	" E.J.	34	50501	Fibroids	"
77	Miss M.D.	29	5246	Do.	"
78	Mrs. M.D.	36	5285	Chron. subinvolution	"
79	" S.M.	32	5323	Do.	"
80	Miss S.W.	40	5854	" Metritis	"
81	" J.K.	34	5949	Metritis	"
82	Mrs. S.R.	42	5970	Mult. Fibroids	"

1936

83	Miss E.M.	30	6155	Chron. Metritis	"
84	" B.J.	27	6222	Chron. Endometritis	"
85/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Number in Hospital Records</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
85	Miss H.B.	36	6503	Fibroid	Nil
86	Mrs. E.W.	35	6513	Do.	"
87	" E.T.	34	6592	Chron. Metritis	"
88	" S.C.	36	7179	Fibroid	"
89	" S.S.	39	1079	Chron. Metritis	"
90	" M.McG.	43	7318	Mult. Fibroids	"

1937

91	Mrs. R.F.	41	7345	Mult. Fibroids	"
92	" J.McC.	38	4330	Chron. Metritis	"
93	" E.McL.	44	7734	Chron. subinvolution	"
94	" E.K.	45	7754	" Metritis	"
95	" L.C.	41	7784	Fibroid	"
96	" E.W.	38	7810	Mult. Fibroids	"
97	" J.C.	43	7818	Fibroid	"
98	" S.S.	33	642	Fibroids	"
99	" J.McL.	33	8226	Mult. Fibroids	"
100	" I.T.	48	4993	Chron. subinvolution	"
101	" I.G.	38	8302	Mult. Fibroids	"

1938

102	" E.McE.	39	8495	Chron. subinvolution	"
103	" E.P.	38	8582	Fibroid	"
104	Miss E.B.	31	8668	Fibroids	"
105	Mrs. J.McG.	42	9009	"	"
106	Miss J.B.	37	9014	Mult. Fibroids	"
107	Mrs. M.K.	42	9018	Fibroid	"
108	" M.V.	39	9088	Mult. Fibroids	"
109	" A.S.	31	9141	Fibroid	"
110	" A.E.	31	9157	"	"
111	Miss L.McN.	31	9268	"	"
112	Mrs. H.C.	39	9342	Single submuc. Polyp.	"
113	" M.J.	38	8270	Chron. subinvolution	"
114	" M.R.	40	9463	Fibroid	"
115	" D.D.	41	9334	Mult. Fibroids	"

1939

116	Mrs. M.M.	43	9794	Cervical Fibroid	"
117	" A.B.	44	9734	Fibroid	"
118	" B.McN.	37	9765	Fibroids	"
119/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Number in Hospital Records</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
119	Mrs. E.B.	42	9787	Fibroids	Nil
120	" M.H.	38	9862	"	"
121	" M.M.	35	9923	"	"
122	" C.D.	36	9472	Chron. subinvolution	"
123	" J.J.	55	10034	Fibroids	"
124	" K.K.	43	1742	Mult. Fibroids	"
125	" E.S.	38	10070	Fibroids	"
126	" M.O'R.	37	10115	Chron. Endometritis	"
127	" A.McD.	43	10242	Do.	"
128	" A.W.	37	10275	Fibroid	"
129	" M.O.	40	10330	"	"
130	" H.McL.	51	1400	"	"
131	" J.McL.	34	8867	Metropathia Haem.	"
132	" C.P.	38	10508	Fibroid	"
133	" M.L.	40	10548	Fibroids	"

1940

134	Mrs. H.P.	53	9613	Fibroid	"
135	" H.L.	32	10890	Fibroids	"
136	" A.S.	46	10932	"	"
137	" C.S.	40	10934	Cystic Glandular Hyperplasia	"
138	" E.K.	38	11066	Mult. Fibroids	"
139	" A.O'N.	34	10191	Hypertrophy of Uterus	"
140	" L.F.	37	10697	Chron. Inflamm.	"
141	" M.M.	39	11379	Fibroids	"
142	" M.W.	44	11409	"	"
143	" M.S.	33	11408	"	"
144	" C.F.	54	11560	Endometrial Poly.	"
145	" M.T.	37	11174	Fibroids	"

1941

146	" W.E.	39	11919	Fibroids	"
147	" E.McM.	46	11941	"	"
148	" M.J.	41	11965	"	"
149	" S.S.	41	12006	"	"
150	" J.W.	41	12147	"	"
151	" M.McK.	38	12328	Adenomyosis	"
152	" E.N.	49	12259	Fibroids	"
153	" E.V.	39	6448	Chron. subinvolution	"
154	" M.P.	32	12107	Metropathia Haem.	"
155	" J.S.	39	12825	Fibroid	"
156	" G.A.	36	12611	Cystic Glandular Hyperplasia	"

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Number in Hospital Records</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
157	Mrs. C.B.	43	12621	Fibroids	Nil
158	" F.A.	42	5374	"	"
159	" J.G.	43	12721	"	"
160	" C.Y.	34	12722	Adenomyoma	"
161	" M.M.	33	13185	Chron. subinvolution	"
162	" C.S.	40	13231	Fibroids	"
<u>1942</u>					
163	" J.J.	39	13232	Fibroid	"
164	" E.D.	43	13265	Fibroids	"
165	" E.B.	37	13335A	"	"
166	" M.P.	42	13263	Cystic Glandular Hyperplasia	"
167	" M.D.	41	13409	Chron. subinvolution	"
168	" M.S.	34	13301	Fibroids	"
<u>1943</u>					
169	Mrs. F.P.	51	13500	Fibroids	"
170	" M.W.	42	13678	"	"
171	" R.G.	50	12962	"	"
172	" L.S.	37	15027	Mult. Fibroids	"
173	" A.P.	43	14981	Do.	"
174	Miss M.E.	37	14935	Fibroids	"
175	Mrs. E.H.	50	14849	Fibroids	"
176	" G.W.	39	14070	"	"
177	" M.P.	34	14048	"	"
178	" B.C.	40	14040	"	"
179	" S.P.	48	14016	"	"
<u>1944</u>					
180	" A.M.	46	15791	Fibroids	"
181	" J.B.	46	15523	Fibroid	"
182	" J.S.	26	15502	Chron. subinvolution	"
183	" S.W.	40	14715	Adenomyoma of Uterus	"
184	" E.B.	50	15657	Fibroids	"
185	" W.M.	39	9221	"	"
186	" M.J.	51	10678	"	"
187	" E.T.	42	15026	Fibroid	"
188	" A.M.	31	13919	Fibroids	"

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Number in Hospital Records</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
<u>1945</u>					
189	Mrs. A.B.	37	7963	Fibroids	Nil
190	" R.F.	39	16145	"	"
191	" A.U.	38	16155	"	"
192	" M.S.	45	2647	Adenomyoma of Uterus	"
193	" M.M.	44	15974	Fibroids	"
194	" M.L.	50	16022	"	"
195	" J.P.	41	15954	"	"
196	" A.C.	44	15707	"	"
197	" J.B.	37	4162	"	"
198	" H.McW.	37	16799	"	"
<u>1946</u>					
199	Mrs. E.S.	65	18168	Fibroids	"
200	" J.G.	43	18065	"	"
201	" M.B.	39	17628	"	"
202	" S.S.	49	17167	"	"
203	" C.A.	43	17026	Fibroid	"
204	" E.K.	36	16860	"	"
205	" R.D.	43	16600	Metropathia Haem.	"
206	" W.D.	40	16715	Chron. subinvolution	"
207	" J.W.	42	16688	Fibroid	"
208	" M.M.	42	16688	Fibroids	"
<u>1947</u>					
209	" C.W.	44	19530	Fibroids	"
210	" F.B.	42	19313	"	"
211	" C.M.	23	19399	"	"
212	" C.D.	41	11400	"	"
213	" J.B.	45	15567	Functional Ut.Haem.	"
214	" E. McL.	36	8453	Cervical Fibroid	"
215	" M.F.	41	13376	Fibroids	"
216	" M.Y.	29	12934	Functional Ut.Haem	Op. for Foll. Cyst - 8/1/54.
217	Miss M.W.	40	19022	Fibroids	Nil
218	Mrs. W.W.	38	19045	"	"
219	" M.S.	43	18859	"	"
220	" J.L.	41	18720	"	"
221	" R.B.	46	18617	"	"
222	" L.K.	43	18476	"	"
223	" C.C.	44	18277	Mucous Polyp.Fibroids	"

Vaginal Hysterectomy (with conservation of Ovaries)

1929 - 1945

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Number in Hospital Records</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
224	Mrs. M.C.	61	1046	Fibroids & Prolapse	Nil
225	" M.W.	60	1134	Chron. Cervitis & Prolapse	"
226	" E.B.	52	1174	Chron. Metritis &	"
227	" J.R.	43	1338	Chron. subinvolution	"
228	" E.L.	47	1999	Do.	"
229	" M.S.	47	2093	Do.	"
230	" M.S.	54	2158	Chron. Metritis	"
231	" M.McG.	36	2620	Do.	"
232	" M.W.	59	3545	Prolapse	"
233	" N.D.	42	3055	Chron. Metritis	"
234	" J.C.	46	5152	Fibroids	"
235	" M.B.	44	6175	"	"
236	" C.C.	60	6673	Chron. Endometritis	"
237	" S.H.	45	7229	Fibroids	"
238	" A.H.	52	7260	"	"
239	" A.H.	27	7743	"	"
240	" M.G.	33	16013	Submuc. Fibroids	"

Dr. D. MacIntyre's Total Hysterectomies with Conservation  
of Ovaries 1928-1946

241	Mrs. A.T.	32	424	Cervical Fibroids	"
242	" E.L.	40	852	Mult. Fibroids	"
243	" J.H.	38	1332	Fibroids	"
244	" M.S.	39	1383	Chron. subinvolution	"
245	" J.S.	38	10013	Cervical Fibroids	"
246	" M.R.	30	12274A	Do. Fibroid	"

Dr. D. MacIntyre's Subtotal Hysterectomies and Unilateral  
Salpingo-oophorectomy 1927

247	Mrs. J.R.	35	9	Fibroid	"
248	" C.McD.	42	10	" +cystic ovary(L)	"
249	" M.B.	45	15	" + "	"
250	" B.McA.	43	2 24	Chron.subinvol. & cystic ovary (L)	"

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Number in Hospital Records</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
<u>1928</u>					
251	Mrs. R.B.	48	189	Polyp. + cys.ov.(L)	Nil
252	" R.B.	43	222	Endometritis + cyst.ov.(L)	"
253	" I.F.	44	257	Chron. Metritis + cys.ov.(R)	"
254	" M.C.	25	260	T.B.Endometritis + salp.(R)	"
255	" M.H.	42	289	Chron. Metritis + ooph.(R)	"
256	" B.K.	39	292	Fibs. + cyst.ov. (L)	"
257	" A.R.	40	356	Chron. Metritis + ooph.(R)	"
258	" E.S.	30	418	Chron. Metritis + " (L)	"
259	" M.I.	36	562	Do. + cyst.ov(L)	"
<u>1929</u>					
260	Mrs. M.McL.	43	401	Chron. Endom. + salp.ooph.(R)	"
261	" J.G.	37	645	Fib. + luteal cyst (L)	"
262	Miss M.B.	25	652	T.B. Endometritis	"
263	Mrs. A.McD.	38	732	Chron. subin. + salp.ooph.(L)	"
264	" M.D.	34	736	Chron.Met. + cyst. ov.(L)	"
265	" A.A.	42	747	Fibs. + cyst. ov. (L)	"
266	Miss J.W.	42	825	Fibs. + foll.cyst. (L)	"
267	" C.S.	37	847	Chron. Met. + salp.(L)	"
268	Mrs. A.E.	37	108	Do. Do.	"
<u>1930</u>					
269	Mrs. M.M.	33	1083	Chron.Met. + Salp.ooph.(R)	"
270	" J.S.	37	1187	Do. (L)	"
271	Miss A.H.	43	1193	Fibs. + Retention Cyst (L)	"
272	Mrs. A.G.	38	1281	Fibs. + cyst.ovary (R)	"
273	" E.L.	35	1330	Chron.subinvol. + cys.ov.(L)	"
274	Miss J.S.	43	1407	Cyst.Gland. Hyperplasia	"
275	Mrs. M.M.	43	1418	Do.	"
276	" J.R.	51	1538	Chron. Metritis	"
<u>1931</u>					
277	Mrs. E.S.	39	1579	Fibs. + Dermoid cyst (L)	"
278	" C.H.	42	1582	" serous cyst (R)	"
279	" A.S.	33	1630	Chron. Met. + Foll.cyst (L)	"
280	" E.E.	42	1646	Fibs. + ooph. (L)	"
281	" A.D.	47	1660	Chron.Met. + cyst.ov. (R)	"
282	" J.W.	25	1802	Do. + ooph. (R)	"
283	" J.C.	37	1834	Do. + Tubal Preg.(L)	"
284	" J.S.	42	1897	Do. + lut.cyst (L)	"
285/					



<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Number in Hospital Records</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
285	Mrs. M.B.	41	1957	Cyst.Gland. Hyperplasia	Nil
286	Miss E.B.	42	1958	" "	"
				+ Fimbrial Cyst (L)	"
287	Mrs. A.W.	41	2025	Chron. Lut. & Cyst. Ov.(R)	"
288	" G.G.	44	2033	Do. Do.	"
289	" M.McA.	41	2052	Fibs. + cyst.ovary (L)	"
290	" H.B.	42	2081	Chron. subinvolution	"
<u>1932</u>					
291	Mrs. M.M.	37	2013	Fibs. + Lut.cyst. (L)	"
292	" A.C.	46	2164	Fibs. + salp.ooph. (L)	"
293	" A.H.	42	2291	Fibroids	"
294	" C.R.	33	729	Fibs. + cyst.ovary (R)	"
295	Miss E. McF.	42	2495	Fibroids	"
296	Mrs. E.H.	27	2563	Chron. Met. + salp.ooph.(R)	"
297	" G.R.	42	2596	Fibroids	"
298	" A.C.	48	2351	Adenomyosis	"
299	" B.L.	34	2641	Fibroids	"
<u>1933</u>					
300	" D.McL.	42	2672	Fibs. + cyst.ov. (L)	9 months later simple cyst of Rt.Ovary
301	" A.C.	42	2712	Fibroids	Nil
302	" J.W.	37	2749	Chron. Metritis	"
303	" L.P.	50	2752	Fibs. + cyst. Ov. (R)	"
304	" M.N.	40	2757	Fibs. + cyst. Ov. (L)	"
305	" H.T.	46	2775	Do. (R)	"
306	Miss J.C.	42	2790	Fibroids	"
307	Mrs. R.McM.	41	2807	"	"
308	" J.S.	38	2814	Fibs. + serous cyst (R)	"
309	" J.I.	41	2906	Fibs. cyst. ov. (R)	"
310	Miss C.D.	38	2973	Fibs. + Pseudomuc. cyst. (L)	"
311	Mrs. M.C.	30	3065	Cyst. Gland.hyperplasia + lut. cyst. (R)	"
312	" M.W.	32	3066	Fibroid	"
313	" E.C.	46	3078	Chron. Metritis	"
314	" A.G.	31	2680	Chron. Met. + cyst. Ov. (R)	"
315	" B.C.	40	3228	Fibs. + cystic Ov. (R)	"
316	" I.M.	25	3237	Fibs. + foll. cyst (R)	"
317	" J.T.	36	3251	Chron.Subinvol.+ cyst. Ov.(R)	"
318	" M.C.	37	3283	Fibs. + simple cyst Ov. (R)	"

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Number in Hospital Records</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
319	Mrs. M.R.	34	3290	Chron. Metritis + cys.ov. (R)	Nil
320	" J.W.	37	3293	Fibs. + cyst.ov. (L)	"
321	" I.Y.	37	3445	Fibs. + cyst.ov. (L)	"
322	" K.B.	38	3529	Chron.subinvol. + Hydrosalpinx (L)	"
323	" M.B.	43	3555	Fibs.+ lutein cyst (R)	"
<u>1934</u>					
324	" H.H.	42	3834	Fibroids	"
325	" A.H.	39	3883	"	"
326	Miss B.R.	31	3914	"	"
327	Mrs. E.S.	33	3991	"	"
328	" F.C.	36	3613	Chron.Met. + cys.ov.(R)	"
329	" H.B.	43	4453	Fibs.oophoritis (R)	"
330	" M.M.	40	4509	Fibroids	"
331	" E.C.	28	2779	Chron. subinvol.	"
332	" A.G.	33	4660	Adenom.+ serous cyst.ov.(R)	"
333	" E.D.	36	4661	" cyst.ov. (L)	"
334	" M.S.	38	3931	Fibroid	"
335	" H.S.	23	3961	Pelvic Tuberculosis	"
336	" M.D.	47	5049	Chron. subinvol.	"
337	" M.I.	39	2181	Fibs. + Rt.foll.cyst (R)	"
338	" I.E.	31	4943	Chron. Endometritis	Cystic Rt. Ovary excised 1936
<u>1935</u>					
339	" E.G.	34	5470	Chron. Metrit. + cyst. ov. (R)	Nil
340	" C.C.	37	2764	Chron. subinvol.	"
341	" M.M.	38	4393	Chron. Endomet.+ cyst.ov(R)	"
342	" A.T.	45	5509	Fibroids	"
343	" M.M.	39	4490	Chron. subinvol. + serous cyst(L)	"
344	" A.B.	45	5648	Fibs. + cyst.ov. (L)	"
345	" J.R.	43	5664	Fibs. + lut. cyst (R)	"
346	" E.D.	36	5681	Cyst.gland hyperplasia + foll.cyst (L)	"
347	" I.L.	35	5726	Chron. Metrit.+ follcyst. (L)	"
348	Miss J.T.	39	5747	Fibs. + Fibroma of ov.(R)	"
349	Mrs. I.S.	40	5791	Fibroids	"
350	" L.K.	39	5840	Fibs. + foll.cyst (L)	"
351/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Number in Hospital Records</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
351	Mrs. J.D.	42	5900	Fib. + foll.cyst (L)	Nil
352	" A.A.	42	5914	Fibs. + cyst.ov. (R)	"
353	" H.McK.	38	5929	Do. (L)	"
354	Miss J.R.	42	5948	Do. (L)	"
355	Mrs. S.T.	38	6012	Fibs. + foll.cyst (L)	"

1936

356	" N.M.	39	6092	Cyst.gland.hyperplasia + foll.cyst (R)	"
357	" M.McC.	41	6113	Chron.endomet.+ foll. cyst (L)	"
358	" C.D.	26	6140	Chron. endomet.+ foll. cyst (R)	"
359	" M.McQ	43	6192	Fibs. + lut.cyst (R)	"
360	" M.H.	41	6220	Fibs. + foll.cyst (L)	"
361	" M.A.	39	6221	Do. (L)	"
362	Miss J.W.	38	6231	Fibs. + foll.cyst (R)	"
363	Mrs. J.McD.	39	6291	Do.	"
364	" M.B.	34	6294	Fib. + Tub.Preg. (R)	"
365	Miss C.C.	37	6432	Fibs. + follic.cyst (L)	"
366	Mrs. M.S.	40	6549	Do.	"
367	" M.M.	38	6550	Adenomyosis	"
368	" E.R.	41	6568	Fib. + cyst.ov. (R)	"
369	" E.W.	41	6587	Do. (L)	"
370	" M.S.	44	6671	Pelvic. Tuberculosis	"
371	" J.B.	46	7058	Fibroid	"
372	" M.McD.	41	7085	Fib. + cyst.Ov. (R)	"
373	" I.C.	44	7120	Adenomyosis	"
374	" J.B.	36	7171	Chron. Endometritis	"
375	" J.H.	46	7191	Adenom. + cyst.ov. (L)	"

1937

376	" A.L.	43	7362	Fibroids	"
377	Miss H.McC.	41	7390	Fibs. + cyst.Ov. (R)	"
378	Mrs. G.B.	48	7540	Fibroid	"
379	" J.F.	40	7587	" + corpus luteum (R)	"

Hysterectomy and Unilateral Oophorectomy

1937 (cont'd)

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Number in Hospital Records</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
380	Mrs. J.C.	37	7702	Fibs. + foll.cyst (L)	Nil
381	" I.A.	42	7745	Fibroids	"
382	Miss A.McG.	38	7746	Fibs. + Haem. of Ov.(L)	"
383	Mrs. J.J.	50	7794	Chron. Endometritis	"
384	" M.M.	50	7866	Chron. Met. + lut.cyst(L)	"
385	" I.P.	38	7918	Fibs. + Fibroma of Ov.(R)	"
386	" I.V.	45	8024	Fibs. + foll.cyst.Ov.(R)	"
387	" L.H.	45	8124	Fib. + cystic Ov. (R)	"
388	" A.M.	44	8185	Fibroid	"
389	" J.C.	43	8235	" + foll.cyst (R)	"

1938

390	" J.McC	42	8251	Fib. + lut.cyst (L)	"
391	Miss M.M.	46	8275	Fibroids	"
392	Mrs. J.W.	31	8486	" + single cyst (R)	"
393	" L.R.	45	8753	Chron. Met. + lut.cyst (L)	"
394	" R.B.	43	8779	Chron. subinvol. + simple cyst (L)	"
395	" I.T.	48	8864	Mult. Fibs. + fibroma of ovary (R)	"
396	Miss J.W.	37	8933	Mult. Fibroids	"
397	Mrs. I.H.	40	8969	Fib. + simple cyst of ov. (L)	"
398	" M.D.	51	9023	Fib. + simple cyst of ov.(L)	"
399	" M.W.	36	9156	Chron. subinvol. + simple cyst of Ov. (L)	"
400	" M.J.	45	9190	Fibroids	"
401	" E.W.	48	9187	Fibs. + foll.cyst. ov. (L)	"
402	" J.S.	42	9201	Fibs. + simple cyst.ov.(R)	"
403	Miss B.F.	30	9184	Fibroids	"
404	" M.McE.	38	9283	Fibs. + simple cyst.ov.(L)	"
405	" M.P.	32	9385	Endomet. of ov. (L)	"
406	Mrs. S.B.	45	9329	Fibroids	"
407	" J.L.	49	9318	Fibs. + foll.cyst.ov.(L)	"
408	" C.C.	35	9410	Fib. + simple cyst.ov.(L)	"
409	" J.T.	43	9452	Fib. + Do.	"
410	" E.P.	44	9542	Fib. + corpus luteum (L)	"

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Number in Hospital Records</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
<u>1939</u>					
411	Mrs. E.G.	36	9534	Chron. Subinvol. + corpus luteum (R)	Nil
412	" A.I.	49	9564	Fibs. + corpus luteum (R)	"
413	" M.B.	34	4311	Chron. subinvol.	"
414	" M.C.	46	9641	Fibroids	"
415	" R.W.	35	9008	Fib. + lut.cyst.Ov(R)	"
416	" M.W.	40	9758	Fibs. + foll.cyst.ov(R)	"
417	" J.W.	37	8950	Cyst.gland.hyper. + simple cyst Ov.(L)	"
418	" E.R.	38	9913	Fibroid	"
419	" A.S.	40	9282	Chron.subinvol. + lut. cyst.Ov. (R)	"
420	" J.N.	35	9992	Adenomy. of Uterus	"
421	" B.G.	38	9994	Chron. subinvol. + lut. cyst (L)	"
422	" C.M.	38	10047	Fib. + corpus.lut. (R)	"
423	" E.D.	39	10053	Fibroids	"
424	" U.B.	46	10145	Metropathia Haem.	"
425	" E.R.	41	10148	Fibroids	"
426	" J. McF.	38	10316	" + simple cyst of L. Ov.	"
427	" J.A.	41	10308	Fibroids	"
428	" J.C.	45	10414	Fibs. + Endom. of L.Ov.	"
429	Miss M.A.	37	10433	Fibroids	"
<u>1940</u>					
430	Mrs. J.W.	46	10660	Fibroid	"
431	" B.B.	41	10721	"	"
432	Miss M.R.	38	10819	"	"
433	Mrs. B.G.	44	11072	Fibs. + corpus lut.(L)	"
434	" M.H.	41	11082	Fibroids	"
435	" M.McG	42	11125	Sub.invol.	"
436	" I.H.	51	11114	Cyst.gland.Hyper. + foll.cyst (R)	"
437	" J.McK.	45	11149	Fibs. + foll.cyst.Ov(R)	"
438	" A.H.	43	11244	Fibroid	"
439	" B.W.	48	11297	Fibs. + simple cyst of L.Ovary	"
440	" M.M.	39	11365	Fibroids	"
441	" E.McD	44	11637	Fibs. + foll.cyst (L)	"
442	" J.K.	45	11693	Do. (R)	"
443	" A.B.	44	4104	Fibroids	"

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Number in Hospital Records</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
444	Mrs. A.F.	43	11882	Fibroids	Nil
445	" P.R.	42	11503	Fibs. + Chron.Salpingo- oophoritis (L)	"
<u>1941</u>					
446	Mrs. E.McL	44	12002	Fibroids	"
447	" M.R.	44	12320	Metropathia + foll. cyst (L)	"
448	" M.W.	39	12315	Fibroids	"
449	" E.B.	37	12532	Metropath.Haem. + foll.cyst (L)	"
450	" R.H.	42	12531	Fibroid	"
451	" C.B.	39	12596	Fibs. + endomet.Ov.(R)	"
452	" C.McN	46	12740	Fib. + fibroma of Ov.(R)	"
453	" M.C.	46	12654	Fibroids	"
454	" A.C.	48	11969	Metropath. Haem.	"
455	" A.W.	47	12685	Mult.Fibroids	"
<u>1942</u>					
456	" F.P.	51	13500	Fibs. + endomet.Ov.(L)	"
457	Miss J.W.	44	13338	Fibroids	"
458	" S.M.	47	13190	Fibroid	"
459	Mrs. M.C.	38	13187	Fib. + lut.cyst (L)	"
460	" G.R.	39	13655	Cervical fibroid	"
461	" S.L.	40	13698	Fibroid	"
<u>1943</u>					
462	" J.McL.	39	13924	Fibroid	"
463	" M.D.	42	13883	Fib. + corpus luetum (L)	"
464	" J.S.	43	13895	Fibroid	"
465	" E.C.	47	14041	Fib. + foll.cyst. Ov.(L)	"
466	" J.G.	43	14109	Fibroids	"
467	Miss R.McF	43	14261	"	"
468	Mrs. J.R.	37	14209	Metropath. Haem.	"
469	" M.McC	41	14285	Fib. + corpus lut.(L)	Pseudomucinosi cyst. R.Ov. Oct.55
470	" I.F.	49	14776	Fibroid	Nil
471	" D.F.	43	14773	Fibroids	"
<u>1944</u>					
472	" G.B.	44	14980	Fib. + pseudomuc.cyst	"
473	" C.D.	41	15114	Fibroids	"
474	" A.B.	38	15153	Fibroid	"
475	Miss A.S.	42	15391	"	"

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Number in Hospital Records</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
476	Mrs. H.McG	37	15487	Fibs. + chron. salp. oophoritis (R)	Nil
477	" J.S.	48	15340	Fibs. + Endomet. Ov.(L)	"
478	Miss L.W.	34	15499	Fib. + foll.cyst.Ov.(R)	"
479	Mrs. M.L.	48	15764	Fibroid	"
480	" W.F.	41	15713	Fibroids	"
481	" A.L.	41	15736	"	"
482	Miss C.F.	40	15766	"	"
<u>1945</u>					
483	Mrs. J.A.	44	16091	Fibroids	"
484	Miss M.McL	30	16136	Fib. + L.salp.oophoritis	"
485	" M.K.	40	16443	Fib. + foll.cyst.Ov.(R)	"
486	Mrs. J.A.	47	16577	Metropath. Haem + foll. cyst.Ov (L)	"
487	Miss J.R.	45	16633	Mult.Fibroids	"
488	Mrs. A.Y.	41	16879	Fibs. + foll.cyst.Ov.(L)	"
489	Miss L.S.	40	16870	Fib. + Endomet. Ov.(R)	"
490	Mrs. A.A.	49	17159	Fibroid	"
491	" J.McF	43	17140	"	"
<u>1946</u>					
492	" M.D.	45	17819	Hydatidiform Mole	"
493	" J.G.	43	18065	Cerv.Fib. + foll.cyst. Ov. (R)	"
494	" J.P.	44	6020	Fib. + foll.cyst. of Ov.(R)	"
495	" I.B.	39	18146	Fibroid	"
496	" J.C.	32	18150	Fibs. + foll.cyst of Ov.(L)	"
497	" L.W.	42	18178	Cyst.gland.Hyperplasia + lut.cyst Ov.(L)	"
498	Miss B.McN	40	18358	Fib. + foll.cyst (R)	"
499	Mrs. A.D.	42	12586	Fib. + chron.Salp. (L)	"
500	Miss C.H.	37	17526	Pelvic. Endomet.	"
<u>1947</u>					
501	Mrs. S.M.	39	18494	Cerv. Fibroid	"
502	" M.D.	41	18519	Fibs. + foll.cyst Ov.(R)	"
503	" C.S.	55	18626	Fibs. + lut.cyst (R)	"
504	" A.T.	45	11598	Mult. Fibroids	"
505	" J.S.	41	19089	"	"
506	" M.R.	58	19244	Fibs. + ser.cystad.(L)	"
507/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Number in Hospital Records</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
507	Mrs. I.A.	40	19533	Fibroids	"
508	" J.F.	43	19558	"	"
509	" E.G.	39	15729	Adenomyosis of Uterus	"
<u>1948</u>					
510	" J.P.	50	20022	Fibroids	"
511	" S.McI	40	20064	"	"
512	" M.R.	45	20139	Fibs. + Pseudomuc. cyst. Ov. (L)	"
513	" B.R.	45	20307	Fibs. + foll.cyst Ov.(R)	"
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Cases of Hysterectomy with Conservation of One  
or Both Ovaries (all units)

SAMARITAN HOSPITAL

1948 - 1955

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinical No.</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
514	Mrs. M.G.	47	12579	Fibroids	Nil
515	Miss A.B.	42	3213	Fibs. + foll.cyst	"
516	Mrs. S.H.	35	20364	Fibroids	"
517	" M.McC	37	5385	"	"
518	" M.I.	39	388	"	"
519	" J.McL	38	2279	Endometritis	"
520	Miss J.B.	34	5413	Fibroid	"
521	" F.C.	50	5485	"	"
522	Mrs. M.S.	40	12730	"	"
523	" E.D.	54	5490	"	"
524	Miss M.C.	50	4304	Fib. + corp.luteum	"
525	Mrs. M.T.	32	12781	Fibroids	"
526	" M.W.	36	12421	Cyst.gland.hyperplasia	"
527	Miss A.T.	31	20521	Endometritis	"
528	" J.McL	45	5530	Fibs. + foll.cyst	"
529	Mrs. M.G.	39	12792	Fibroids	"
530	" M.L.	33	15238	"	"
531	" M.A.	51	11579	Adenomyosis	"
532	" M.McL	23	12835	Pelv. T.B.	"
533	" M.B.	43		Cyst.gland.hyperplasia	"
534	" M.McS	40	8109	"	"
535	" C.E.	39	5588	Fibroids	"
536	" M.G.	44	12851	"	"
537	" H.A.	24	17894	No abnormality	"
538	" M.M.	36	12953	Fibroid	"
539	" M.McC	50	12931	"	"
540	" C.B.	42	12945	No abnormality	"
541	" A.M.	38	6635	Adenomyosis	"
542	" M.H.	33	4952	No abnormality	"
543	" C.M.	39	20635	Fibroids	"
544	Miss E.M.	40	20800	Cyst.gland. Hyperplasia	"
545	Mrs. M.McT	44	5676	Fibroids	"
546	" M.G.	38	472	"	"
547	" A.McK	37	17199	Fib. + simple ov.cyst	"
548	" E.B.	39	13445	No abnormality	"
549	" H.McF	49	6308	Fibroid	"
550	" M.J.	45	13652	"	"
551/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinical No.</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
551	Mrs. M.C.	49	13680	Fibroids	Nil
552	Miss M.S.	45	13704	"	"
553	Mrs. M.C.	51	13689	"	"
554	" M.H.	35	13699	"	"
555	Miss E. McW	38	6373	"	"
556	Mrs. M.B.	55	13716	Endomet. polyp.	"
557	" M.I.	65	563 Pr.	Chron. cervitis	"
558	Miss M.S.	32	13755	Fibroid	"
559	Mrs. M.I.	45	13770	"	"
560	" M.H.	44	136 Pr.	"	"
561	" M.S.	52	13754	"	"
562	" M. McC	44	10343	"	"
563	" M. McC	49	6424	Hyperplasia of endomet.	"
564	" A.M.	44	6455	Fibroids	"
565	" M.H.	52	13769	Chron. endomet.	"
566	" M.W.	35	2900	No abnormality	"
567	" Y.B.	43	6489	Hyperplasia of endomet.	"
568	" M.N.	31	13860	"	"
569	" M.N.	40	6483	Fibroid	"
570	" M.G.	44	13841	"	"
571	" M.W.	48	13047	"	"

1949

572	" A.C.	33	11958	Hyperplasia of endomet.	"
573	" M.F.	47	13180	Fibroid	"
574	" M.S.	44	13187	"	"
575	" M. McD	43	13189	Fibroids	"
576	" M.R.	36	13195	"	"
577	" J.A.	45	13196	"	"
578	" C. McM	50	13188	"	"
579	" M.B.	53	13183	Adenomy. + simple cyst	"
580	" A.S.	49	13191	Fibroids	"
581	" A.W.	39	13216	Fibs. + foll. cyst	"
582	" E.C.	43	13206	Fibroid	"
583	" M.W.	43	5901	"	"
584	" M. McK	43	13229	"	"
585	" M.D.	73		Fibroid	"
586	" R.B.	45	13231	Fibs. + hydrosalpinx	"
587	" H. McB	47	13113	" + lut. cyst	"
588	" M. McG	45	916	Fibroids	"
589	" A.W.	33	20853	"	"
590	" S.B.	47	5244	Fibs. + corp. luteum	"
591/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinical No.</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
591	Mrs. M.H.	39	6424	No abnormality	Nil
592	" M.E.	48	13209	Fibroids	"
593	" J.M.	43	13289	"	"
594	" M.S.	45	5957	"	"
595	" M.G.	40	6024	Adenomyosis	"
596	" M.O.	44	6000	Fibroids	"
597	" E.P.	49	13321	Fib. + chron.salp.	"
598	" M. McL	45	13389	" + foll.cyst	"
599	" E.G.	51	Pr.5113	No abnormality	"
600	" A.W.	38	6046	Fib. + endomet.	"
601	" M.B.	47	6051	Fibs. + normal ov.	"
602	" A.S.	48	13376	Hyperplasia of endomet.	"
603	" A.B.	42	13385	Fibs. + foll.cyst	"
604	" M.W.	43	13474	No abnormality	"
605	" A.McK	35	1355	Ut. + foll.cyst	"
606	" M.S.		1340	Endomet.hyperplasia	"
607	" A.R.	41	13403	Fibs. + foll.cysts.	"
608	" E.M.	42	13417	Fibroids	"
609	" E.B.	41	6091	Cyst.gland.hyperplasia	"
610	" E.P.	68	21206	Fibs. + serous cyst	"
611	" C.W.	43	6100	" + normal ov.	"
612	" A.D.	44	6087	Fibroids	"
613	" A.C.	44	13489	"	"
614	" J.M.	42	21389	" + corp.luteum	"
615	" E.M.	38	10578	" "	"
616	" C.M.	67	6204	"	"
617	" B.O'R	41	6181	" "	"
618	" H.W.	51	6191	"	"
619	Miss K.F.	46	Priv.	"	"
620	Mrs. M.A.	45	858	Fib. + corp. luteum	"
621	" M.B.	38	13528	Endomet.hyperplasia	"
622	" H.M.	51	13574	No abnormality	"
623	" M.McG	50	20820	Fibroids	"
624	Miss M.H.	41	13591	Fibs. + T.B.salp.ooph.	"
625	" E.P.	38	13577	Fibroids	"
626	" M.M.	45	13563	"	"
627	Mrs. A.G.	33	18026	No abnormality	"
628	" M.B.	41	13604	Fibroids	"
629	Miss I.S.	39	3968	Fibs. + foll.cyst	"
630	Mrs. M.B.	46	13899	Fibroid	"
631	" M.D.	47	13594	"	"
632	" M.B.	41	13664	"	"
633	" M.F.	43	21552	" polyp.	"

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinical No.</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
<u>1950</u>					
634	Mrs. A.McL	35	22034	Fibroid	Nil
635	E.F.	40	6535	Adenomyosis	"
636	M.F.	46	3903	Fibroid	"
637	M.A.	52	13921	No abnormality	"
638	M.G.	34	13940	Fibroid	"
639	C.S.	35	189	"	"
640	Miss J.M.	45	5309	Hyperplasia of endomet.	"
641	Mrs. A.M.	46	13980	Fibroid	"
642	L.B.	48	2124	"	"
643	Miss M.McL	36	193	"	"
644	Mrs. M.McI	35	13988	Endometritis	"
645	C.L.	37	22095	Fibroid	"
646	A.S.	40	9316	Fib. + lut.cyst	"
647	E.R.	45	14032	Fibroid	"
648	H.C.	35	4389	"	"
649	Miss .A.D.	47	274	Cyst.gland.hyperplasia	"
650	Mrs. A.G.	37	13767	Fibroid	"
651	M.M.	44	22159	Fib. + foll.cyst	"
652	M.G.	42	6626	"	"
653	Miss L.R.	53	14085	Hyperplasia of endomet.	"
654	Mrs. M.J.	42	19050	Fibroids	"
655	M.A.	47	13137	"	"
656	M.P.	46	5506	Cyst.gland.hyperplasia	"
657	J.S.	43	2579	"	"
658	E.W.	49	Pr.233	Hyperplasia of endomet.	"
659	Miss M.S.	33	20813	Cyst.gland.hyperplasia	"
660	Mrs. M.B.	39	22193	Fibroid	"
661	E.C.	40	21646	Cyst.gland.hyperplasia	"
662	M.T.	52	8227	Fibroid	"
663	Miss W.K.	44	22280	Fibroid	"
664	Mrs. J.L.	43	14186	Fib. + foll.cyst	"
665	M.C.	51	14196	Adenomyosis	"
666	J.McI	43	14175	Fibroids	"
667	M.McF	47	22288	Polypoidal endomet.	"
668	W.E.	44	14202	Fibroids	"
669	M.B.	49	14185	"	"
670	Miss A.B.	49	14209	"	"
671	G.B.	42	14245	"	"
672	Mrs. M.G.	51	14247	"	"
673	A.C.	36	Priv.	Simple cyst	"
674	L.S.	52	14221	Fibroid	"
675	R.S.	42	14223	"	"
676	M.R.	35	2828	No abnormality	"
677/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinical No.</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
677	Mrs. M.H.	46	11605	Fibroid	Nil
678	" F.M.	43	6849	"	"
679	" M.M.	30	6524	Fimb.cyst	"
680	" M.H. McK	42	6524	Fibroid	"
681	" E.S.	44	6889	No abnormality	"
682	Miss I. McC	58	22497	Fibroid	"
683	Mrs. C.L.	41	6929	"	"
684	" M.H.	38	14311	No abnormality	"
685	" C.L.	42	Priv.	Fibroids	"
686	" E.C.	51	14336	"	"
687	" M.S.	46	6928	Fib. + foll.cyst	"
688	" M.G.	43	Pr.305	Fibroids	"
689	Miss M.L.	41	22628	Fibroid	"
690	Mrs. M.B.	36	14412	Fib. + foll.cyst	"
691	" M.K.	42	7022	Fibroid	"
692	" I.C.	36	7036	"	"
693	" M.T.	48	22644	"	"
694	" E.S.	45	Pr.630	Fib. + fimb.cyst	"
695	" J.R.	39	14464	Fib. + simple cyst	"
696	" M.L.	39	6764	Hydrosalpinx	"
697	" I.S.	48	14473	Fibroid	"
698	" J.D.	50	14515	Fib. + ser.cyst	"
699	" S. McC	49	14490	Fibroid	"
700	" L.K.	48	7136	"	"
701	" J.G.	37	7140	Polyp. + corp.luteum	"
702	" E.C.	38	13369	Metropath.Haem.	"
703	" M.S.	55	1075	Fib. + ser.cyst	"
704	" E.S.	50	14530	Fibroids	"
705	" M.M.	36	5720	Fibroid	"
706	" E.L.	47	14584	"	"
707	Miss E. McD	42	14578	Endomet. of ov.	"
708	Mrs. R.R.	46	14576	Fibroid	"
709	" E.M.	48	22585	"	"
710	" M.C.	40	7219	"	"
711	" A.S.	36	7211	"	"
712	" A.B.	40	14625	No abnormality	"
713	" A.G.	42	21858	Hyperplasia of endomet.	"
714	" M.B.	39	21649	Fibroid	"
715	" E.C.	43	14638	"	"
716	" M.D.	51	23009	"	"
717	" M. McD	43	7254	"	"
718	" J.G.	51	14616	"	"
719	" M. McM	45	14666	"	"
720	" M.C.	32	7772	Cyst.gland.hyperplasia	"
721	" E.T.	41	14680	Chron.endomet.	"
722	" M.B.	43	14654	No abnormality	"
723	" C.D.	46	12599	Cyst.gland.hyperplasia	"
724/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinical No.</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
724	Mrs. C.H.	40	14721	Fibroid	Nil
725	" M.W.	42	14513	Adenomyosis	"
726	" M.McC	40	7295	Endomet.hyperplasia	"
727	" M.A.	39	34213	"	"
728	Miss J.S.	31	14707	Fibroid	"
729	" M.L.	26	13636	Endomet.hyperplasia	"
730	Mrs. M.Y.	39	7354	"	"
731	Miss M.W.	43	7359	Fibroid	"
732	" M.McK	44	14737	"	"
733	Mrs. M.B.	43	7357	"	"
<u>1951</u>					
734	Mrs. A.McC	46	14770	Fibroid	"
735	" I.D.	28	6801	"	"
736	" M.M.	38	23253	"	"
737	" M.B.	45	7388	"	"
738	" M.D.	38	13929	Endomet.hyperplasia + lut.cyst	"
739	" J.S.	45	14839	Fibroid	"
740	" A.N.	45	14817	Adenomy. + lut.cyst	"
741	" M.D.	50	4089	Fibroid	"
742	Miss M.H.	50	14824	"	"
743	" M.C.	38	23357	"	"
744	Mrs. M.L.	41	14280	Endomet.hyperplas.+ foll.cyst	"
745	" C.R.	40	14875	Endomet.hyperplasia	"
746	" S.A.	43	7507	Hyperpl. endometrium	"
747	" M.H.	34	18674	"	"
748	" M.F.	40	23410	Prolif. of endomet.	"
749	" M.G.	50	7505	"	"
750	" K.McK	48	14936	"	"
751	Mrs. J.McL	46	14908	"	"
752	" J.G.	43	14555	Endomet.hyperplasia	"
753	Miss I.F.	47	Priv.	Fibroid	"
754	" A.S.	29	Priv.	Lut.cyst	"
755	Mrs. D.McF	47	14930	Cyst.gland.hyperplasia	"
756	" M.H.	46	23577	Fib. + foll.cyst	"
757	" A.M.	66	14947	Fibs. + simple cysts	"
758	" J.K.	42	5693	Fib. + lut.cyst	"
759	" J.S.	41	14947	Fibroid	"
760	" A.M.	52	15001	"	"
761	" M.McT	49	7612	"	"
762	" M.R.	56	14994	"	"
763	" M.M.	43	11792	"	"
764	" M.E.	46	15044	"	"
765	" M.E.	37	20373	Endomet.hyperplasia	"
766/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinical No.</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
766	Mrs. A.R.	44	7640	Cyst.gland.hyperplasia	Nil
767	" M.C.	47	15133	Fibroid	"
768	" M.McC	43	15092	Myohyperplasia	"
769	" M.McP	48	15110	Endomet.hyperplasia	"
770	" P.W.	49	15021	Fibroid	"
771	" A.R.	41	7720	"	"
772	" M.A.	46	15200	Fibs. + endomet. of ov.	"
773	Miss J.McQ	45	15192	Fibroid	"
774	Mrs. E.H.	45	7759	"	"
775	" G.B.	33	15265	Adenomyosis	"
776	Mrs. M.D.	71	15309	Chron. inflam.	"
777	" C.S.	39	6095	Endometritis	"
778	Miss A.B.	39	7533	Endomet.hyperplasia	"
779	" A.S.	38	7823	Fibroids	"
780	Miss M.E.	32	15325	Fibs. + simple cyst	"
781	Mrs. M.M.	46	23978	Ut.normal + simple cyst	"
782	Miss M.H.	50	15356	Fibroid	"
783	Mrs. M.M.	44	15375	" + corp.luteum	"
784	" A.K.	51	15403	Fibroids	"
785	" M.McD	63	15408	Fibroid	"
786	" M.G.	34	15421	"	"
787	" A.D.	34	7947	"	"
788	" C.McA	50	15500	"	"
789	" E.B.	35	24258	"	"
790	Miss M.McC	36	8007	"	"
791	Mrs. A.J.	36	15807	"	"
792	" J.G.	46	40813	Endometritis	"
793	" M.W.	37	24337	Fibroid	"
794	" G.G.	46	15396	"	"
795	" A.K.	44	8057	Fib. + lut.cyst	"
796	" M.G.	41	15571	No abnormality	"
797	" M.O'D	41	6001	Fibroid	"
798	" J.D.	31	15578	"	"
799	" H.H.	46	24355	"	"
800	" C.D.	31	8056	Fibroids	"
801	Miss J.A.	44	15053	"	"
802	Mrs. E.R.	44	Pr.322	Fib. + fibroma of ov.	"
803	" H.F.	30	15081	No abnormality	"
804	" N.J.	37	15663	Foll. cysts of ov.	"
805	" J.P.	35	15052	Subinvol. + foll.cyst of ov.	"
806	" C.McF	50	15595	Fibroids	"
807	" J.W.	44	24487	Endomet.	"
808	" A.McG	45	8126	Fibs. + foll.cyst	"
809	" G.F.	52	15458	" + pseudomuc.cyst	"
810	Miss E.A.	48	7989	Fibroids	"
811	Mrs. A.G.	48	15697	"	"
812	Miss M.S.	45	7461	No abnormality	"

813/

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinical</u> <u>No.</u>	<u>Pathology</u>	<u>Re-admission for</u> <u>Ovarian Pathology</u>
813	Mrs. C.S.	44	Pr.245B	Fib. + lut.cyst	Nil
814	" C.McC	43	15685	Fibroids	"
815	" J.E.	43	11148	Simple cyst of ovary	"
816	" M.R.	60	15762	Fibroid	"
<u>1952</u>					
817	Mrs. J.H.	38	15773	Fibroid	"
818	" D.S.	44	15768	Fib. + foll.cyst	"
819	" M.H.	46	15289	" + fimb.cyst	"
820	" J.B.	38	24707	Fibroid	"
821	" A.McF	39	24607	"	"
822	" A.B.	42	15840	Fib. + simple cyst	"
823	" W.H.	44	10486	Myohyperplasia	"
824	" M.C.	39	7524	Endomet. of ovary	"
825	" C.McD	43	8308	Fibroid	"
826	" S.K.	37	1094	"	"
827	" M.D.	42	13125	Endomet.hyperplasia	"
828	Miss M.F.	54	B.48	Fibroid	"
829	Mrs. A.G.	42	8329	" + simple cyst	"
830	" M.McG	44	24843	"	"
831	" A.D.	33	14507	"	"
832	" C.W.	44	Pr.402B	"	"
833	" M.J.	48	" 404B	"	"
834	" E.B.	43	15978	Endomet.hyperplasia	"
835	" M.K.	43	8367	Do. + simple cyst	"
836	Miss M.F.	43	Pr.	Fibroids	"
837	Mrs. M.McL	27	"	"	"
838	Miss M.McK	38	H15765	Fibs. + corp.luteum	"
839	" M.G.	37	8449	Fibroid	"
840	Mrs. G.D.	39	7011	Chron. Endomet.	"
841	" M.C.	42	7041	No abnormality	"
842	" M.R.	39	Pr.B416	Fibs. + endomet.	"
843	Miss E.H.	43	25089	Fibroids	"
844	Mrs. M.M.	46	7456	"	"
845	" M.B.	43	8523	Metropath.haem.	"
846	Miss I.T.	40	20225	Fibroid	"
847	Mrs. C.H.	42	16144	"	"
848	" M.L.	40	Pr.47B	"	"
849	Miss M.M.	41	8577	"	"
850	Mrs. C.B.	29	16185	"	"
851	" D.E.	40	15651	Fib. + corp.luteum	"
852	Miss L.C.	40	988	" + foll.cyst	"
853	Mrs. A.L.	39	8610	No abnormality	"
854	" K.C.	37	15293	Cyst.gland.hyperplasia	"
855	" S.T.	23	7843	Myohyperplasia	"
856/					



<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinical No.</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
857	Mrs. M.W.	36	6364	No abnormality	Nil
858	" M.G.	41	8636	Myohyperp. + simple cyst	"
859	" E.H.	47	8653	Metropath.Haem.	"
860	" I.M.	39	27270	Fibroid	" "
861	" N.T.	48	25445	"	"
862	Miss M.R.	38	14526	Polyp.	"
863	Mrs. M.G.	43	25491	Foll.cyst of ov.	"
864	" R.S.	67	25465	Fibroid	"
865	" J.K.	37	8745	" + foll.cysts	"
866	" B.McA	47	8746	"	"
867	" M.T.	57	Pr.434	"	"
868	" J.G.	51	8782	Fibroids	"
869	" C.D.	42	8778	Fibroid	"
870	" J.N.	45	25543	"	"
871	" M.P.	50	16332	"	"
872	" R.McE	46	7609	Fibs. + fibroma of ov.	"
873	" M.McC	43	25568	Fibroid	"
874	" M.F.	38	25569	Polyp.	"
875	" M.C.	38	8818	Fib. + foll.cyst	"
876	" A.D.	39	16381	Fibroids	"
877	" B.R.	47	8820	"	"
878	" A.R.	38	16406	"	"
879	" B.C.	36	25647	" + corp.lut.	"
880	" M.G.	31	25469	Fibroid	"
881	" E.F.	51	8862	Fib. + ser.cyst.	"
882	" M.T.	49	25697	Fibroid	"
883	" M.D.	41	8885	"	"
884	" M.C.	34	16299	Cyst.gland.hyperp.	"
885	" J.McF	44	25777	Fibs. + corp.lut.	"
886	Miss M.H.	43	6197	Fibroids	"
887	Mrs. M.McD	38	16525	Fibroid	"
888	" A.T.	55	8900	No abnormality	"
889	" M.McK	43	16561	Fibs. + simple cyst ov.	"
890	Miss D.S.	40	16583	Fibroids	"
891	Mrs. S.P.	48	16577	Fibroid	"
892	" M.M.	42	8865	Cyst.gland.hyperplasia	"
893	" R.S.	38	6434	Fibroid	"

1953

894	" H.G.	36	16569	Polyp.	"
895	" K.W.	41	15959	Fibroid	"
896	" C.R.	46	9003	Fibroids	"
897	" N.H.	49	9018	Fibroid	"
898	" M.D.	42	16997	"	"
899	" M.W.	43	Priv.	Corp.lut.	"

1000/

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinical No.</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
1000	Mrs. S.B.	43	25983	Fibroid	Nil
1001	" J.H.	46	17058	Adenomy.Uteri	"
1002	" A.D.	37	25728	Myohyperplasia	"
1003	" M.W.	63	17047	Fibroids	"
1004	" M.C.	39	13227	Endomet.hyperplasia	"
1005	Miss M.A.	39	4059	"	"
1006	Mrs. J.J.	48	17031	Fibroid	"
1007	" J.C.	49	Pr.458	" + endomet.	"
1008	Mrs. F.A.	41	17122	"	"
1009	" H.M.	38	17009	"	"
1010	Miss J.G.	49	Pr.245	Fibs. + Pseudomuc.cyst	"
1011	Mrs. J.T.	41	17160	Fibroids	"
1012	" E.A.	41	10035	Adenomyosis	"
1013	" J.I.	39	Pr.164	Endomet.hyperplasia	"
1014	" M.W.	44	14610	No abnormality	"
1015	" M.G.	42	9169	Fibroid	"
1016	Miss A.McD	51	9190	"	"
1017	" C.G.	47	28403	Endomet.hyperplasia	"
1018	Mrs. E.T.	41	17264	Fibroid	"
1019	Miss K.C.	28	26192	Fibs. + simple ov.cyst	"
1020	Mrs. E.McA	39	9270	Fibs. + foll.cyst	"
1021	" E.A.	45	17299	Fibroid	"
1022	" G.G.	36	12332	No abnormality	"
1023	" E.D.	47	9314	Fibroid	"
1024	" M.T.	39	9346	"	"
1025	" M.S.	50	Pr.327	"	"
1026	" J.C.	29	18669	"	"
1027	" M.S.	45	22528	"	"
1028	" C.C.	40	17208	"	"
1029	" M.T.	37	5320	No abnormality	"
1030	" M.R.	39	17382	Fibroids	"
1031	Miss M.McK	43	15856	"	"
1032	Mrs. J.McL	39	17446	Myohyperplasia	"
1033	" M.M.	45	17480	Fibs. + endomet.	"
1034	" M.G.	47	17462	Fibroids	"
1035	" J.B.	34	14836	No abnormality	"
1036	Miss A.W.	48	26614	Fibroids	"
1037	Mrs. K.F.	38	17438	"	"
1038	Miss I.M.	40	17472	Fibs. + foll.cyst	"
1039	Mrs. I.McV	49	17508	Fibroid	"
1040	" J.D.	42	26641	Fib. + corp.lut.	"
1041	Miss M.B.	26	17550	Fibroid	"
1042	Mrs. M.C.	38	Priv.	Fibs. + foll.cysts	"
1043	" E.M.	39	175814	"	"
1044	" M.W.	39	15789	Haem. of broad lig.	"
1045/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinical No.</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
1045	Mrs. L.D.	47	16425	adenomyosis	Nil
1046	" J.R.	41	9516	Fibroids	"
1047	" R.G.	53	17593	"	"
1048	" A.H.	44	17588	Foll.cyst of ov.	"
1049	" A.C.	39	17417	No abnormality	"
1050	Miss J.G.	49	17642	Fibroids	"
1051	Mrs. F.F.	49	17369	Fibs. + foll.cysts	"
1052	Miss .C.B.	42	17688	Fibroids	"
1053	Mrs. A.P.	58	Priv.	Adenomyosis	"
1054	" M.K.	39	17671	Fib. + endomet.	"
1055	" E.D.	43	17638	Fibroid	"
1056	" A.H.	49	17695	"	"
1057	Miss M.A.	49	25005	Fib. + hydrosalp.	"
1058	Mrs. H.R.	42	2798	Fibroid	"
1059	" G.R.	42	17505	Metropath.haem.	"
1060	" R.McF	29	26441	Fibroid	"
1061	Miss J.B.	49	9707	"	"
1062	Mrs. E.H.	49	Priv.	"	"
1063	" J.C.	60	17804	"	"
1064	Miss H.F.	45	26643	Fibroids	"

1954

1065	Mrs. B.D.	40	27005	Simple cyst of ov.	"
1066	Miss S.J.	37	9572	Fibroid	"
1067	Mrs. C.L.	50	27069	Adenomyosis	"
1068	Miss M.B.	40	Priv.	Fibroid	"
1069	Mrs. A.B.	54	9719	Cyst.gland.hyperplasia	"
1070	Mrs. R.D.	41	26980	Fibroid	"
1071	Miss J.P.	38	25367	No abnormality	"
1072	Mrs. G.M.	50	9805	Fibroid	"
1073	" E.D.	36	15620	Fib. polyp. + corp.lut.	"
1074	" M.O'B	51	9825	Fibroid	"
1075	" M.M.	46	9841	Fib. + simple cyst	"
1076	" E.R.	48	Pr.612	Fibroid	"
1077	" A.L.	37	12683	No abnormality	"
1078	" A.M.	39	17693	Endomet.hyperplasia	"
1079	" E.E.	43	8544	Corpus luteum	"
1080	" C.A.	43	14225	Fibs. + endomet.	"
1081	" M.T.	46	18006	Simple cyst.of ov.	"
1082	" A.A.	41	6381	Cervitis	"
1083	" M.W.	38	9549	No abnormality	"
1084	" E.P.	45	27292	"	"
1085	" H.C.	46	27308	Mult.Fibroids	"
1086	" M.McF	66	27270	Fibroids	"
1087	" M.S.	35	9002	Salpingitis	"
1088/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinical No.</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
1088	Mrs. M.A.	41	27331	Fibs. + corp.lut.	Nil
1089	" H.O.	43	17954	Fibs. + dermoid cyst	"
1090	" A.McC	41	9883	Fibroid	"
1091	Miss I.L.	49	18016	"	"
1092	Mrs. M.R.	46	18060	Fibroids	"
1093	" J.D.	34	15961	Fibs. + foll.cyst	"
1094	" B.D.	29	25935	No abnormality	"
1095	" M.J.	35	Priv.	Corp.luteum	"
1096	" E.W.	48	9941	Hydrosalpinx	"
1097	" M.J.	35	9957	Fibroids	"
1098	" S.McN	49	9976	"	"
1099	" C.P.	49	8693	Fibroid	"
1100	" J.B.	51	5861	"	"
1101	" J.M.	41	9985	Hydrosalpinx	"
1102	" J.F.	48	18152	Fibroid	"
1103	" C.H.	49	18116	Fib. + endomet.	"
1104	Miss M.S.	40	9994	Fib. + corp.luteum	"
1105	" E.D.	50	27532	Fib. + fibroma	"
1106	Mrs. J.L.	38	27241	Fibroid	"
1107	" M.M.	43	27534	"	"
1108	" C.N.	34	9245	No abnormality	"
1109	" M.B.	45	9710	Fib. polyp.	"
1110	" E.G.	46	10020	Hyperplasia of endomet.	"
1111	" F.R.	46	10031	Fibroid	"
1112	" M.M.	38	17707	Foll.cyst	"
1113	" H.M.	35	9369	Polyp.	"
1114	" E.G.	35	10081	Fibroid	"
1115	" A.B.	40	10067	"	"
1116	" J.B.	38	7313	No abnormality	"
1117	" J.R.	45	18250	Endometriosis	"
1118	" E.T.	34	27712	Fibroid	"
1119	Miss M.K.	47	27709	"	"
1120	Mrs. M.R.	38	10117	"	"
1121	" K.R.	44	21881	"	"
1122	" E.B.	54	Priv.	No abnormality	"
1123	" E.T.	41	9792	Foll.cyst of ov.	"
1124	" C.S.	41	27785	Fibroid	"
1125	" K.R.	38	27741	"	"
1126	" R.S.	38	18622	Endometriosis	"
1127	" S.B.	47	9088	Cyst.gland.hyperplasia	"
1128	" A.G.	32	10163	Fibroid	"
1129	" M.McC	40	18352	"	"
1130	" A.E.	41	18366	"	"
1131	Mrs. M.S.	30	Priv.	Pyosalpinx	"
1132	" A.B.	46	10209	Fibroids	"
1133/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinical No.</u>	<u>Pathology</u>	<u>Re-admission for Ovarian Pathology</u>
1133	Mrs. H.M.	52	10276	Fibroid	Nil
1134	" A.G.	44	10236	Cyst.gland.hyperplasia	"
1135	" A.B.	39	14849	Fibroids	"
1136	" E.C.	46	10303	Fib. + foll.cyst	"
1137	" A.D.	46	18441	Fib. + ser.cyst	"
1138	" M.McC	47	18489	Fibroids	"
1139	" H.C.	37	17809	Fibs. + corp.lut.	"
1140	" E.S.	45	9072	Aænomy. of uterus	"
1141	" L.B.	38	28030	Endomet.hyperplasia	"
1142	" J.McG	31	27678	Fibroid	"
1143	" M.M.	43	18510	"	"
1144	Miss M.McF	45	18579	"	"
1145	Mrs. B.F.	44	10117	Hydrosalpinx	"
1146	" I.McI	44	10334	Fibroid	"
1147	" H.S.	46	18498	Fib. + foll.cyst	"
1148	" E.McN	44	18522	Fib. + foll.cyst	"
1149	" M.W.	42	10059	Cyst.gland.hyperplasia	"
1150	" A.B.	44	9469	Fibroid	"
1151	" E.C.	46	18555	Fib. + foll.cyst	"
1152	" M.D.	41	Pr.	Fibroid	"
1153	" F.C.	41	10398	No abnormality	"
1154	" M.A.	48	Pr.	Fibroid	"
1155	" M.K.	42	10361	"	"
1156	" V.F.	39	13937	No abnormality	"
1157	" B.S.	32	27949	Fibroids	"
1158	" J.R.	40	10466	"	"
1159	" E.M.	42	10480	"	"
1160	" E.W.	41	10463	Cyst.gland.hyperplasia	"
1161	" P.G.	38	5221	Endomet.	"
1162	" H.D.	40	18716	Fibroid	"
1163	" L.S.	36	18718	Fibroids	"
1164	" J.H.	41	18705	Fibs. + hydrosalp.	"
1165	Miss M.M.	30	14012	No abnormality	"

1955

1166	Mrs. N.C.	31	28085	No abnormality	"
1167	" H.I.	40	17036	"	"
1168	Miss E.M.	40	10592	Fibroid	"
1169	Mrs. M.N.	41	23953	Foll.cyst	"
1170	" J.F.	40	10611	No abnormality	"
1171	" G.S.	66	28075	Fibroids	"
1172	" F.F.	43	18902	"	"
1173	" H.W.	46	10676	"	"
1174	" M.McC	42	18950	Polyp.	"
1175	" A.A.	37	28197	Endomet.	"
1176/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinical</u> <u>No.</u>	<u>Pathology</u>	<u>Re-admission for</u> <u>Ovarian Pathology</u>
1176	Mrs. M.A.	45	10691	Foll.cyst	Nil
1177	" M.H.	55	18960	Fibroids	"
1178	" M.C.	42	28684	"	"
1179	" J.B.	36	28628	Fibs. + foll.cyst.	"
1180	" A.G.	44	10753	Polyp. + foll.cyst.	"
1181	" D.L.	42	10761	Fibroid	"
1182	" A.S.	44	9296	No abnormality	"
1183	" M.F.	56	28689	"	"
1184	Miss M.G.	62	8735	No report	"
1185	Mrs. E.D.	48	Pr.	"	"
1186	" M.W.	34	19057	Fibroids	"
1187	" A.S.	33	18588	No report	"
1188	" R.W.	44	2615	Fibroids	"
1189	" R.B.	41	6342	Cyst.gland.hyperplasia	"
1190	" A.A.	41	18621	Fibs. + papill.ser.cyst	"
1191	" M.W.	39	18880	No report	"
1192	" S.C.	37	10841	Fibroid	"
1193	" J.L.	51	26895	Fibroids	"
1194	" J.W.	48	19126	"	"
1195	" M.C.	47	19099	"	"
1196	" A.M.	34	18884	"	"
1197	" I.F.	46	19137	"	"
1198	" J.B.	54	10970	"	"
1199	" A.C.	46	Prv.	"	"
1200	" M.B.	36	29114	Fibs. + chron.salp.	"
1201	" K.W.	48	10992	Fibs. + simple ser.cyst	"
1202	" I.H.	52	11014	Endomet.hyperplasia	"
1203	" A.A.	37	11034	Foll.cyst	"
1204	" F.S.	39	11067	Fib. + ser.cyst	"
1205	" M.B.	43	11047		"
1206	Miss J.S.	42	11093	Fibroids	"
1207	Mrs. H.U.	68	29308	No abnormality	"
1208	" M.F.	47	11107	Fibroids	"
1209	" A.G.	40	29435	"	"
1210	" E.F.	41	19396	"	"
1211	" J.C.	40	30042	"	"
1212	" E.McK	53	Pr.	"	"
1213	" M.M.	36	19467	"	"
1214	" M.McL	36	28740	"	"
1215	" M.M.	32	19457	"	"

Cases of Oophorectomy 1927-1948 (Dr. McIntyre's List)

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Record Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
<u>1927</u>					
1	Mrs. I.S.	27	46	Cystic ovary (L)	No
2	" M.McN	28	48	" (R)	"
3	" A.McC	30	54	" (L)	"
4	Miss E.McA	50	71	Pseudomuc.cyst (R)	"
5	Mrs. M.A.	40	83	Carcinoma of R.Ov.	"
6	" A.C.	31	164	Dermoid (L)	"
<u>1928</u>					
7	" M.F.	23	195	Cystic Ov. (L)	"
8	" C.C.	45	825	" (R)	"
9	" M.L.	50	251	Solid carcinoma of Ov.(L)	"
10	" C.G.	28	412	Cyst. Ov. (R)	"
11	" A.A.	42	492	" (L)	"
12	" J.G.	58	532	Pseudomuc.cyst (R)	"
13	" A.P.	38	696	Salp.oophor.(R)	"
<u>1929</u>					
14	Mrs. C.K.	29	213	Ser.cyst (R)	"
15	" S.V.	23	731	Salp.oophor. (R)	"
16	" J.B.	32	911	Endomet. (R)	"
17	" K.C.	28	978	T.B.Salp.Oophor.(L)	"
18	" I.G.	29	990	Lut.cyst (R)	"
19	" J.C.	29	1040	Salp.oopho.(L)	"
<u>1930</u>					
20	" M.M.	37	1099	Pseudomuc.cyst (R)	"
21	" E.McK	46	1085	Cyst.Ovary (L)	"
22	" E.S.	38	1262	Foll.cyst (R)	"
23	" E.I.	45	1390	Multi.cyst. (L)	"
24	" S.M.	38	1437	Retention cyst (R)	"
25	" J.R.	38	1499	Foll.cyst (L)	"
26	" J.A.	40	1520	Pseudomuc.cyst (L)	"
<u>1931</u>					
27	Miss E.L.	54	1620	Dermoid (L)	"
28	Mrs. H.A.	64	1690	Fibroma of Ov. (L)	"
29/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Record Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
29	Mrs. M.A.	29	1791	Pseudomuc. cyst (L)	No
30	Miss L.J.	26	1822	" (L)	"
31	Mrs. E.G.	36	1828	Dermoid (L)	"
32	" A.H.	27	1972	Papill.ser.cyst (L)	"
33	Miss C.N.	28	2024	2 Dermoids (R & L)	"

1932

34	Mrs. A.K.	42	2082	Oophor. (L)	"
35	" I.A.	29	2106	Lut.cyst (R)	"
36	" G.F.	27	2135	Simple cyst (R)	"
37	" M.McC	32	2457	Salp.ooph. (L)	"

1933

38	Mrs. J.W.	39	2651	Dermoid (R)	"
39	" H.K.	32	2863	Pseudomuc. (R)	"
40	" M.R.	34	2930	Terat.cyst (L)	"
41	Miss M.C.	19	3040	Pseudomuc. cyst (R)	"
42	" J.T.	28	3047	" (L)	"
43	Mrs. A.C.	19	3053	Simple cyst (L)	"
44	Miss M.McL	60	3180	Pseudomuc. cyst (L)	"
45	Mrs. J.E.	33	3236	Ser.cyst (R)	"
46	" M.M.	28	3467	Ser.cyst (L)	"
47	Miss A.B.	55	3544	Ser.cysts (R & L)	"

1934

48	Mrs. M.T.	42	3618	Ser.cyst (L)	"
49	" J.McK	29	3107	Oophor. (L)	"
50	" A.B.	34	4178	Simple cyst (L)	"
51	" M.McG	21	4663	Foll.cyst (L)	"
52	" A.B.	34	5639	Dermoid (R)	"
53	" M.T.	34	4186	Oophor. (L)	"
54	" E.E.	37	4199	Ser.cyst (L)	"

1935

55	" M.R.	38	4403	Lut.cyst (L)	"
56	" E.P.	55	5216	Pseudomuc. cyst (L)	"
57	" J.M.	32	5228	Foll.cyst (L)	"
58	" D.B.	27	5320	"	"
59	" M.S.	30	5361	Endomet. (L)	"



<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Record Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
<u>1936</u>					
60	Mrs. H.F.	30	6103	Lut.cyst (R)	No
61	" H.B.	27	6437	Ser.cyst (L)	"
62	" E.P.	32	6640	Lut.cyst (R)	"
63	" J.McC	40	6670	Dermoid (L)	"
64	" A.A.	25	7257	" (R)	"
<u>1937</u>					
65	" E.I.	33	7303	Lut.cyst (L)	"
66	" A.M.	32	7660	" (R)	"
67	" C.McK	45	7682	Ser.cyst (R)	"
68	" E.W.	57	7711	Cyst. ov (R)	"
69	" R.McK	31	8011	Endomet. (L)	"
70	" A.S.	72	8125	Fibroma (R)	"
71	Miss N.C.	19	8294	Ser.cyst (R)	"
72	Mrs. A.B.	32	8319	Foll.cyst (R)	"
73	" J.T.	45	8369	Dermoid (R)	"
74	" A.A.	53	8449	Torsion of ov. (L)	"
<u>1938</u>					
75	" A.McC	63	8459	Papil. ser.cyst (R)	"
76	Miss R.McD	35	8549	Pseudomuc. cyst (R)	"
77	Mrs. R.G.	47	8569	" (R)	"
78	" G.D.	29	8630	Ser.cyst (R)	"
79	" M.R.	28	4779	Lut.cyst (R)	"
80	" M.M.	37	8763	Torsion of Ov. (L)	"
81	" M.A.	34	8986	Fibroma (R)	"
82	" A.McM	37	9011	Pseudomuc. cyst (R)	"
83	" P.E.	27	9053	" (L)	"
84	" C.McR	39	9059	Ov. Preg. (L)	"
85	" M.McA	31	9119	Dermoid (L)	"
86	" C.G.	29	9337	" (R)	"
87	" K.McC	28	8338	Ser.cyst	"
<u>1939</u>					
88	" J.T.	38	9692	Fibroma (R)	"
89	" M.L.	66	10063	T.B. Oophor. (R)	"
90	" E.McL	29	10402	Gran.cell tum. (R)	"
91	" M.McL	38	8525	Lut.cyst (R)	"

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Record Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
<u>1940</u>					
92	Miss E.McC	26	10559	Ser.cyst (L)	"
93	" M.M.	35	10799	Endomet. (R)	"
94	" J.C.	26	11040	Ser.cyst (R)	"
95	" M.H.	40	3142	" (L)	"
96	Mrs. I.McG	24	11016	Abcess of ov. (R)	"
97	Miss B.A.	39	11045	Lut.cyst (R)	"
98	Mrs. E.McN	30	11228	Dermoid (L)	"
99	" J.B.	41	11288	" (R)	"
100	" E.R.	33	11431	Pseudomuc. cyst (R)	"
<u>1941</u>					
101	" J.L.	29	11771	Dermoid (R)	"
102	" J.R.	40	11863	Endomet. (L)	"
103	" J.McL	73	12072	Fibroma (L)	"
104	Miss M.W.	25	12394	Foll.cyst (R)	"
105	Mrs. M.T.	33	12562	Endomet. (R)	"
<u>1942</u>					
106	" A.S.	25	12791	Lut.cyst (R)	"
107	" K.F.	42	12930	Simple cyst (R)	"
108	" E.Y.	58	13424	Ser.cyst (R)	"
<u>1943</u>					
109	" M.McL	46	13859	Simple cyst (L)	"
110	" C.G.	22	13969	Endomet. (R)	"
111	" E.U.	35	14044	Foll.cyst (L)	"
112	" L.A.	36	14243	" (R)	"
<u>1944</u>					
113	" J.S.	36	15823	Dermoid (R)	"
114	" A.M.	30	15530	Foll.cyst (L)	"
115	" M.D.	38	15714	Lut.cyst (L)	"
116	" A.I.	29	15673	Foll.cyst (R)	"
117	" R.McC	21	15827	" (L)	"
118	" M.C.	22	16691	" (L)	"
<u>1945</u>					
119	" M.R.	19	17638	Pseudomuc. cyst (L)	"
<u>1946</u>					
120	Mrs. J.P.	33	17614	Pseudomuc. cyst (R)	"

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Record Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
121	Mrs. G.M.	53	17742	Ser.cyst (L)	No
122	" J.McG	36	11730	Pseudomuc .cysts 2 (R) and (L)	"
123	" A.McF	56	17488	Brenner (L)	"
124	" C.S.	25	18037	Pseudomuc.cyst. (L)	"
125	" E.T.	40	18090	Simple cyst (R)	"
126	Miss C.M.	23	18093	Simple ser.cyst (L)	"
127	" J.G.	31	18142	Ser.cyst (R)	"
128	" H.M.	45	18106	" (R)	"

1947

129	Mrs.A.M.	37	17245	Foll.cyst (L)	"
130	" A.McD	37	18810	Pseudomuc.cyst (L)	"
131	" D.R.	40	18876	Torsion.cyst	"
132	" H.McA	30	19462	Dermoid (L)	"
133	" J.W.	23	19461	Simple cyst	"
134	" A.McV	63	19502	Fibroma of ov. (L)	"
135	" J.F.	35	19527	Ovarian cyst (L)	"

Resection of Ovaries

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
<u>1927</u>					
136	Mrs. S.R.	30	44	Simple cystic degen. (L)	Nil
137	Miss A. McC	38	54	Cyst. degen. of ovaries	"
138	Mrs. H.B.	31	122	Simple cyst. degen. (L)	"
<u>1928</u>					
139	Miss M.S.	24	150	Foll. cysts of ovaries	"
140	Mrs. M.A.	34	168	Cyst. degen. of ov. (R)	"
141	" A.W.	26	194	Cyst. ovaries	"
142	" A.C.	42	196	Corp. lut. ov. (R)	"
143	" C.K.	29	213	Ser. cyst. ov. (R)	"
144	" E.J.	39	214	Do.	"
145	" E.L.	24	224	Cystic Ovaries	"
146	" J.G.	28	226	Cys. degen. of ovaries	"
147	" E.M.	30	238	L. salp. ooph.	"
148	" E.D.	30	247	Cyst. ov. (L)	"
149	" M.M.	23	250	cyst. degen. ov. (L)	"
150	" J.L.	23	248	cyst. ov. (L)	"
151	" M. McK	27	323	"	"
152	Miss M.R.	23	381	Cyst. ovaries	"
153	Mrs. M.P.	28	473	Cyst. degen. ov. (L)	"
154	" M.W.	24	480	Cyst. ov. (R)	"
155	" J. McD	43	507	Pseudomuc. cyst (L)	"
156	" W.B.	29	521	Cyst. degen. ov. (L)	"
157	" J.D.	31	547	" ovaries	"
158	" M. McA	32	558	" ov. (L)	"
<u>1929</u>					
159	" M.L.	30	578	Cyst. degen. ovaries	"
160	" E.M.	25	584	Corp. lut. (R)	"
161	" E.C.	30	262	Lut. cyst. (R)	"
162	" S.S.	35	642	Cyst. ov. (L)	"
163	Miss E.T.	19	646	T.B. Salp. ooph. (R) & (L)	"
164	Mrs. M. McC	28	824	Ret. cyst. ov (R)	"
165	" E.R.	30	842	Cyst. degen. ov. (R)	"
166	" S.H.	36	850	" ovaries	"
167	" E. McC	23	893	Cyst. ov (R)	"
168	" A. McC	25	895	"	"
169	" I.G.	33	907	No report	"
170	" J.B.	32	911	Cyst. degen. ov. (R)	"
171	" E.S.	21	944	Lut. cyst (L)	"
172	" S.G.	31	950	Cyst. ov (L)	"
173/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
173	Mrs. J.McE	31	971	Cyst.ovaries	Nil
174	" E.McK	35	295	Cyst. ov (R)	"
175	" I.L.	27	985	No report on ovary	"
176	" V.S.	29	992	"	"
<u>1929</u>					
177	" M.T.	45	994	Cyst. ov. (R)	"
178	Miss G.G.	27	1012	Cyst.degen. of ov. (R)	"
<u>1930</u>					
179	Mrs. C.McD	32	1042	Cyst. ov. (L)	"
180	" J.C.	29	1040	Dyst.degen. ov. (L)	"
181	" A.McD	29	1085	T.B. Salp.ooph.bilat.	"
182	" R.O'C	26	1091	Cyst. ov. (L)	"
183	" A.O.	31	331	" (R)	"
184	" M.F.	35	1095	Fibroma of ov. (L)	"
185	" J.M.	29	1131	Cyst.ov. (R)	"
186	Miss J.W.	23	1170	Cyst.degen. ovaries	"
187	Mrs. M.S.	31	1230	Haema. of ov. (L)	"
188	" E.B.	34	1322	Cyst. ov (R)	"
189	" M.L.	28	1368	"	"
190	" G.H.	34	1380	"	"
191	" M.M.	30	1382	" (L)	"
192	" H.C.	31	1407	Cyst. ov. (R)	"
193	" C.McG	28	1419	Lut.cyst. ov. (R)	"
194	" J.R.	38	1499	Cyst.degen. ov. (L)	"
195	" J.G.	40	1520	Cyst. ov (R)	"
196	" M.McC	33	1542	Lut.cyst. ov. (L)	"
197	" L.R.	35	1551	No report. L. ov. resected.	"
<u>1931</u>					
198	" C.H.	30	1564	Cyst. ov. (L)	"
199	" M.M.	28	1567	Lut.cyst ov. (R)	"
200	" E.S.	39	1578	Dermoid cyst. (L)	"
201	" B.McC	40	1624	Cyst. ov. (L)	"
202	" M.A.	29	1791	Pseudomuc.cyst. (L)	"
203	" H.G.	32	1799	Lut.cyst. (R)	"
204	" L.J.	26	1822	Pseudomuc.cyst. (L)	"
205	" C.K.	30	1865	Cyst. ov. (R)	"
206	" W.R.	37	1870	Resec. ov. R. No report	"
207	" E.V.	36	1878	Cyst. ov.(R)	"
208	" A.S.	35	1938	Cyst. ov. (L)	"
209/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
209	Miss A.L.	29	431	Cyst. ov. (R)	Nil
210	" C.N.	28	2024	Bilat. Terat.cysts	"
<u>1932</u>					
211	Mrs. G.F.	27	2135	Bilat.cyst.ovaries	"
212	" M.R.	25	2192	Foll.cyst. (R)	"
213	" E.L.	26	2256	Lut.cyst. (R)	"
214	" A.C.	48	2350	Bilat.cyst. ovaries	"
215	Miss F.G.	30	2568	Cyst. ov. (L)	"
216	Mrs. G.McN	26	2621	" (R)	"
<u>1933</u>					
217	" J.McI	26	2637	Cyst. ov. (R)	"
218	" M.S.	29	2650	"	"
219	" J.W.	39	2681	Corp.lut. (L)	"
220	" E.McE	30	2663	Bilat.salp.ooph.	"
221	Miss J.S.	23	594	Cyst. ov. (L)	"
222	" S.F.	24	2408	"	"
223	" N.R.	31	2696	Cyst.degen. ov. (R)	"
224	" K.C.	32	2723	Cyst. ov. (R)	"
225	Mrs. I.T.	29	2782	Cyst. ov. (R)	"
226	" E.W.	26	2900	Lut.cyst. (L)	"
227	" M.R.	25	2192	Foll.cyst. ov (L)	"
228	" M.G.	30	3035	Lut.cyst. (L)	"
229	" D.McL	42	2672	Resection. R. ov. no path. report.	Prev.hyst. + L. Salp.ooph. 9 months earlier
230	" J.M.	35	2811	Lut.cyst. of ov.	Nil
231	" E.S.	26	3559	Cyst. ov (R)	"
<u>1934</u>					
232	" R.O'R		3749	Cyst. ov (R)	"
233	" M.M.	28	3789	Lut.cyst. (R)	"
234	" A.T.	38	3797	Cyst. ov. (R)	"
235	" S.M.	38	3908	Foll.cyst. (L)	"
236	" E.B.	23	4115	Ret.cysts. ovaries	"
237	" M.C.	38	4120	Cyst. ov. (R)	"
238	" M.M.	39	4249	Dermoid cyst. ovaries	"
239	" J.D.	26	1906	Bilat.cyst.ovaries	"
240	" Q.D.	43	4706	Cyst. ov. (R)	"
241	" A.G.	43	4707	" (L)	"
242	" H.McC	28	2757	Foll.cyst. ov. (R)	"
243	" J.F.	37	2490	Foll.cyst. l ov.	"
				Lut.cyst. other ov.	"
244	" E.C.	25	5012	Lut.cyst. (L) ov.	"

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
<u>1935</u>					
245	Mrs. H.S.	23	3961	T.B. Bilat.salp.ooph.	Nil
246	" M.M.	33	5213	Cyst. ov. (L)	"
247	" A.D.	23	5339	" (R)	"
248	" E.D.	36	5681	Lut.cyst. ov. (R)	"
249	" J.H.	29	5779	Cyst. ov. (R)	"
250	" F.McP	28	3085	Bilat.cyst. ovaries	"
251	" J.C.	36	58b2	Cyst. ov. (R)	"
<u>1936</u>					
252	" A.McM	43	6275	Lut.cyst. (R)	"
253	" E.W.	36	6339	Cyst. ov. (R)	"
254	Miss M.L.	23	6375	Lut.cyst. (R)	"
255	Mrs. H.D.	19	6418	Lut.cyst. ov. (L) Dermoid cyst (R) ov.	"
256	" J.G.	32	6532	l. foll.cyst. (L) ov. Fibroma ov. (R)	"
257	" E.S.	28	6630	Bilat. ser.cysts	"
258	" G.J.	27	6694	Corp.lut. ov. (L)	"
259	" H.K.	28	6759	Cyst. ov. (R)	"
260	" A.B.	27	7045	Lut.cyst. (L)	"
261	" A.B.	39	7066	Pseudomuc. cyst (L) ov.	"
262	" J.McC	40	6670	Corp.lut. (R) ov. Dermoid cyst (L) ov.	"
263	Miss M.B.	15	6668	Foll.cyst. (L) ov.	"
<u>1937</u>					
264	Mrs. A.F.	35	7285	Corp.lut. (R)	"
265	" J.M.	33	5228	Cyst. ov. (R)	"
266	" M.C.	26	7374	Foll.cyst. (L) ov.	"
267	" M.M.	37	2310	Foll.cyst.s ovaries	"
268	" S.McP	37	7518	Endomet. (L) ov.	"
269	Miss A.O'D	33	7548	Cyst.degen. ovaries	"
270	Mrs. H.J.	33	7607	Cyst. ov. (R)	"
271	" A.H.	28	7995	Lut. cyst. (R) ov.	"
272	" A.M.	25	8084	Ser.cyst. (R) ov.	"
273	" J.H.	30	7890	Corp.lut. (L) ov.	"
274	" J.McC	42	8251	Lut.cyst. (R) ov.	"
275	Miss N.C.	19	8294	Ser.cyst. (R) ov.	"
276	" C.H.	29	7389	Ret. oyst. (L) ov.	"

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
<u>1938</u>					
277	Mrs. J.T.	45	8369	Cyst. ov. (L) Dermoid cyst. (R) ov.	Nil
278	" G.B.	33	8452	Endomet. ov. (R)	"
279	" R.McD	35	8547	Pseudomuc. cyst. (R) ov.	"
280	" R.G.	47	8569	"	"
281	" I.A.	40	8574	Cyst. ov. (L)	"
282	" I.Y.	26	8591	" (R)	"
283	Miss E.C.	25	8636	" (L)	"
284	Mrs. E.S.	31	8821	Endomet.	"
285	" C.McR	39	9059	Ov. Preg.	"
286	" M.McL	30	9127	Pseudomuc. cyst. (R)	"
287	" A.S.	27	9406	Lut.cyst. (L) ov.	"

1939

288	Miss M.B.	35	4949	Endomet. both ovaries	"
289	" J.McQ	27	9999	Pseudomuc. cyst. (L) ov.	"
290	Mrs. M.S.	42	10118	Lut.cyst. (R) ov.	"
291	" C.S.	32	10256	Endomet. both ovaries	"
292	" A.L.	38	10321	Cyst.degen. ovaries	"
293	" C.D.	29	10459	Pseudomuc. cyst. (R) ov.	"

1940

294	" M.B.	45	10568	Pseudomuc. cyst. (L) ov. Cyst. ov. (R)	"
295	" E.McG	34	10582	Endomet. (R) ov.	"
296	" M.D.	26	10646	Ser.cyst. (L) ov.	"
297	" A.McQ	24	10730	Pseudomuc. cyst (L) ov. Cyst. ov (R)	"
298	" E.McL	37	10770	Pseudomuc. cyst (R) ov. Fibroma (L) ov.	"
299	" C.F.	28	10780	Ser.cyst. (R) ov.	"
300	Miss R.J.	21	11112	Corp.lut. (R) ov.	"
301	Mrs. J.W.	34	11335	Pseudomuc. cyst. (L) ov.	"
302	Miss A.B.	28	11429	" (R)	"
303	Mrs. M.C.	33	11510	Tubal preg.	"
304	" J.P.	38	9823	Cyst. ov. (R)	"
305	" M.M.	29	11584	Corp.lut. ov. (R)	"
306	Miss E.S.	25	11634	Ser.cyst. ov. (R)	"

1941

307	Mrs. J.L.	29	11771	Dermoid cyst. ov.	"
308	" E.L.	33	11810	Lut.cyst. (R) ov.	"



<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
309	Mrs. C.M.	31	11798	Lut.cyst. (L) ov.	Nil
310	" M.McL	39	11677	Foll.cyst. (L) ov.	"
311	" M.P.	31	12393	Cyst. ov. (L) Pseudomuc. cyst (R) ov.	"
312	" I.H.	41	12466	"	"
313	" E.B.	37	12532	Cyst. ov. (R)	"
314	Miss B.F.	26	12543	Ser.cysts - ovaries	"
315	Mrs. M.W.	29	12569	Pseudomuc. cyst. (R) ov. Cyst. ov. (L)	"
316	" S.T.	28	12575	"	"
317	Miss E.A.	18	12744	Bilat. ser.cysts	"

1942

318	Miss M.G.	18	12798	Pseudomuc. cyst. (L) ov. Lut.cyst. (R) ov.	"
319	Mrs. M.M.	25	11567	Foll.cysts (R) ov.	"
320	" H.McC	41	12993	Corp.lut. (R) ov.	"
321	Miss A.M.	28	12795	Cyst. ov. (R)	"
322	Mrs. E.W.	32	13206	Foll.cyst. (R) ov.	"
323	" M.L.	24	13240	Ser.cyst. ovaries	"
324	" I.P.	23	13257	Lut.cyst. (L) ov.	"
325	" M.B.	33	13487	Ser.ctst. (R) ov.	"
326	Miss F.S.	21	13605	Lut.cyst. (L) ov.	"

1943

327	Mrs. E.W.	35	14044	Endomet. (R) ov. Foll.cyst. (L) ov.	"
328	Miss E.M.	25	13279	" (R)	"
329	Mrs. R.C.	23	14355	Endomet. (R) ov.	"
330	Miss M.L.	22	14790	Pseudomuc. cyst. (R) ov. Foll. cyst (L) ov.	"

1944

331	Miss I.McG	24	14930	Endomet. of. (L) ov.	"
332	Mrs. L.T.	34	14965	Foll. cyst. (R) ov.	"
333	" R.M.	29	15034	Simple cysts (L) ov.	"
334	" S.D.	41	15219	Ser.cyst. (R) ov.	"
335	" J.L.	31	15324	Foll.cysts. (R) ov.	"
336	" M.M.	31	15307	Degen.corp.lut. (L) ov.	"
337	" A.F.	34	15416	Pseudomuc.cyst. (L) ov.	"
338	Miss A.M.	30	15530	Foll.cysts. ovaries	"
339	Mrs. A.C.	34	15490	Fibroma of (R) ov.	"
340/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
340	Mrs. M. McC	38	15636	Foll. cysts. of (L) ov.	Nil
341	" M.D.	38	15914	Lut. cyst. (L) ov.	"
342	" R. McC	21	15827	Foll. cysts. (R) ov.	"
<u>1945</u>					
343	" M.B.	21	15935A	Foll. cysts. ovaries	"
344	" E.M.	24	16062	Terat. (L) ov.	"
345	" H.S.	27	16055	Foll. cysts. (R) ov.	"
346	Miss R.D.	43	16600	Foll. cyst (R) ov.	"
347	" M.A.	20	16961	Pseudomuc. cyst. (R) ov.	"
<u>1946</u>					
348	Mrs. V.R.	33	17986	Simple cysts. ovaries	"
349	" M.G.	22	16691	Foll. cyst. (R) ov.	"
350	Miss M.C.	36	16920	Foll. cysts (R) ov.	"
351	Mrs. M.B.	30	5397	Foll. cyst (R) ov. Lut. cyst. (L) ov.	"
<u>1947</u>					
352	" E. McE	30	18590	Lut. cyst. (L) ov.	"
353	" J. McK	37	18622	Foll. cyst. (L) ov.	"
354	Miss M.H.	21	18485	Lut. cyst. (R) ov.	"
355	Mrs. A. McK	22	19611	Foll. cysts. (R) ov.	"
<u>1948</u>					
356	" M.H.	35	16151	Simple cysts - ovaries	"
357	" E.B.	27	15841	Endomet. - ovaries	"

Cases of Oophorectomy 1948 - 1955

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinical Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
<u>1948</u>					
358	Mrs. E.C.	30	20381	Ectopic Preg. (L)	Nil
359	" M.L.	33	12638	Endomet. (L)	"
360	" H.S.	32	12669	Endomet. (L)	"
361	" J.McB	24	20477	Cyst. (R) ov.	"
362	" J.McL	27	20515	Foll.cyst. (L)	"
363	" A.McI	40	5510	Ser.cyst. (L)	"
364	" M.C.	26	12768	" (R)	"
365	" A.M.	19	5514	T.B. Salp. (R)	"
366	" M.C.	31	11159	Ectopic (R)	"
367	" C.T.	34	20508	Endomet. (L)	"
368	" M.T.	35	12830	Ser.cyst. (R)	"
369	" J.P.	33	5590	Endomet. (R. & L.)	"
370	" D.T.	25	20569	Tube-ov. abcess (L)	"
371	" M.V.	36	5577	Ectopic Preg. (R)	"
372	" M.H.	40	12874	"	"
373	" M.McC	27	5596	Pseudomuc. cyst (L)	"
374	" M.B.	36	20741	Fibroma (R)	"
				Foll.cyst (L)	"
375	" M.R.	26	12917	T.B. Salp. (R)	"
376	" M.McC	43	20750	T.B. Abcess (L)	"
377	" J.C.	52	5604	Pseudomuc. cysts. (R & L)	"
378	Miss M.S.	27	12948	T.B. Salp. (R. & L.)	"
379	Mrs. M.W.	28	4650	Ser.cyst (R)	"
380	" M.G.	35	12921	Chron.salp.ooph. (R & L)	"
381	" M.J.	26	12929	Ectopic Preg. (L)	"
382	" M.McF	25	20525	Tube-ovarian Abcess (R)	"
383	" M.A.	21	12961	T.B. Salp. (R)	"
384	" E.P.	43	14981	Ser.cysts. adenoma (L)	Hysterectomy 1944
385	" M.C.	69	5972	Ser.cyst (L)	Nil
386	" M.B.	31	20646	Endomet. (L)	"
387	" M.E.	28	16872	T.B. Salp.ooph.	"
388	" C.C.	38	20509	No abnormality (R)	"
389	" M.Y.	77	20454	Papil.cysts (R & L)	"
390	" M.I.	55	12830	Carcinoma of Ov.	"
391	" M.F.	25	5691	Ser.cyst. (R)	"

1949

392	" M.D.	62	13003	Ser.cyst. (R)	"
393	" M.A.	62	13050	Fibroma (R)	"
394/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
394	Miss S.McG	16	20575	Foll.cyst. (R)	Nil
395	Mrs. A.N.	36	20315	Hydrosalpinx. (L)	"
396	" A.McG	32	20835	" (R & L)	"
397	" R.B.	30	20826	Salp. (R)	"
398	" J.N.	28	20824	Lut.cyst. (R)	"
399	" C.T.	64	13022	Brenner (L)	"
400	" A.G.	30	13175	T.B. Salp.ooph. (R & L)	"
401	" J.H.	32	13194	Foll.cysts (R)	"
402	" A.S.	26	13181	Cyst. ov. (R)	"
403	" M.B.	32	20873	Pseudomuc. cyst (L)	"
404	" J.W.	33	13793	Salp.ooph. (R & L)	"
405	Miss E.D.	34	5884	Lut.cyst. (L)	"
406	" V.A.	24	12415	Ser.cyst. (R)	"
407	" M.McR	52	13238	Carcinoma of Ov.(R)	"
408	Mrs. J.T.	28	13211	Foll.cyst (R)	"
409	" J.T.	30	13213	" (L)	"
410	" M.S.	38	21013	Fibroma (R & L)	"
411	" B.McC	38	21059	Fibroma (R)	"
				Foll. cyst (L)	"
412	" M.McL	36	1321	Lut.cyst. (L)	"
413	" J.C.	26	13207	Pseudomuc.cyst. (L)	"
414	" M.S.	64	21096	Gran.cell (R & L)	"
415	" A.McB	37	5978	Tubal Mole	"
416	" E.McR	40	13291	Ser.cyst (R)	"
417	" M.H.	36	21136	Lut.cyst. (L)	"
418	" M.McP	64	1121	Pseudomyc.cyst. (R & L)	"
419	" A.L.	35	13337	Ser.cyst (L)	"
420	" R.S.	43	13388	Lut.cyst (R)	"
421	" E.F.	34	Priv.	Lut.cyst (L)	"
422	" M.R.	24	19086	Foll.cyst (L)	"
				Corp.lutm (R)	"
423	" J.R.	31	13428	T.B. Salp.ooph. (R & L)	"
424	Miss M.B.	32	13402	Pseudomuc. cyst (R)	"
425	Mrs. M.R.	23	13449	Ser.cyst (L)	"
426	" E.McC	24	6091	Carcinoma of Ov.	"
427	" L.F.	58	21262	Pseudomuc. cyst (L)	"
428	" M.W.	58	Priv.	"	"
429	" G.S.	29	6098	Foll.cysts (R & L)	"
430	" F.McG	47	13517	Ser.cyst (R)	"
431	Miss S.O'D	28	12635	Salp.ooph.(L)	"
432	Mrs. J.A.	37	13527	Foll.cyst (L)	"
433	" M.D.	37	6177	Pseudomuc.cyst (L)	"
434	" M.A.	25	Priv.	No abnormality	"
435	" M.S.	31	13519	Pseudomuc.cyst (L)	"
436	Miss S.Y.	36	Priv.	Do.	"
437	Mrs. M.F.	26	21933	Ectopic	"
438/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
438	Mrs. C.F.	26	6255	Lut.cyst.	Nil
439	" J.H.	61	21523	Carcinoma of ov. (L)	"
440	" A.McL	27	21259	Chron.Salp.ooph (R)	"
441	" M.W.	33	6271	Ectopic Preg. (L)	"
442	" E.W.	46	7272	Dermoid (L)	"
443	" M.M.	31	21510	Ser.cyst (L)	"
444	" M.R.	36	13614	Foll.cyst (R)	"
445	" A.D.	34	6299	Carcinoma of ov. (R)	"
446	" M.G.	39	6305	Chron.Salp.ooph. (R)	"
447	" M.B.	29	5229	Ectopic (R)	"
448	" A.C.	46	13660	Ser.cyst (R)	"
449	" M.H.	28	13678	T.B. Salp. (R)	"
450	Miss M.F.	16	13696	Salp. (L)	"
451	Mrs. M.E.	21	21625	" (R)	"
452	" A.McL	51	21551	Pseudomuc.cyst (L)	"
453	" M.McA	59	13741	"	"
454	" M.G.	23	13757	Lut.cyst (R)	"
455	" B.K.	32	21200	Pseudomuc.cyst (R)	"
456	" J.McM	47	6415	Ser.cyst (R)	"
457	" R.P.	21	21725	Foll.cyst (R)	"
458	" M.R.	33	12691	Endomet. (R)	"
459	" C.McC	25	6481	Ectopic	"

1950

460	" C.D.	32	14107	Ser.cysts (R & L)	"
461	" C.L.	41	22257	Simple cysts (R & L)	"
462	" M.L.	25	Pr.216	Dysgerm. (L)	"
463	" M.B.	40	6667	Foll.cyst (L)	"
464	" J.H.	24	22213	Pseudomuc.cyst (R)	"
465	" M.B.	35	14149	" (L)	"
466	" M.Y.	49	22235	Cystadenom. (R)	"
467	" M.McE	33	12845	Foll.cyst (R)	"
468	Miss B.C.	43	2406	Endomet. (R)	"
469	Mrs. A.M.	24	22341	Ser.cystad. (R)	"
470	" C.H.	25	14233	Salp.ooph. (R)	"
471	" M.K.	39	11313	Tub.-Ov. abcess (R & L)	"
472	" J.McG	51	6848	Pseudomuc. cyst (L)	"
473	" M.O'C	22	14275	Lut.cyst (L)	"
474	" C.N.	32	14315	Tubal mole (L)	"
475	" H.R.	25	14300	Endomet. (L)	"
476	" E.L.	37	13212	Ser.cyst (L)	"
477	" M.Q.	51	14327	Pseudomuc. cyst (R)	"
478	" M.McK	42	6942	" cysts (R & L)	"
479	Miss M.McK	19	22558	" cyst (L)	"
480	Mrs. M.McC	57	22574	Gran.cell tumour (L)	"
481/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
481	Miss M.P.	34	22758	Pseudomuc. cyst (L)	Nil
482	Mrs. A.D.	29	22720	Tubal mole (R)	"
483	" H.T.	49	14487	Path.unknown. Ov.cyst (L)	"
484	" F.K.	41	22777	Simple cyst (L)	"
485	" M.McL	49	14535	Pseudomuc. cyst (L)	"
486	" J.D.	50	7126	Ser.cyst (R)	"
487	" M.W.	24	17831	Lut.cyst (L)	"
488	" C.M.	54	14539	Pseudomuc. cyst (L)	"
489	" M.McD	30	14544	Ectopic (L)	"
490	" M.McF	48	22835	Simple cysts (R & L)	"
491	" E.T.	47	14528	Pseudomuc. cysts (R & L)	"
492	Miss M.R.	50	22916	" (R)	"
493	Mrs. E.C.	46	22946	Brenner tum. (L)	"
494	" M.McM	32	22919	Endomet. (R)	"
495	" M.W.	40	14580	Pseudomuc. cysts (R & L)	"
496	" M.L.	36	18344	Lut.cyst (R)	"
497	" M.McI	62	14660	Ser.cyst (R)	"
498	Miss M.S.	34	22984	" (L)	"
499	Mrs. M.M.	28	23048	Salp.ooph. (L)	"
500	Miss M.D.	28	23689	Ser.cystad. (L)	"
501	Mrs. M.D.	24	23689	" (R)	"
502	" M.H.	38	23079	" (L)	"
503	" M.P.	27	23117	Pseudomuc. cyst (L)	"
504	" M.O.	67	7314	" (R)	"

1951

505	" M.B.	50	23190	Pseudomuc. cyst (L)	"
506	Miss M.McC	27	23293	Salp.ooph. (R)	"
507	Mrs. E.H.	29	23279	Ser.cysts (R & L)	"
508	" M.G.	28	13043	Foll.cyst (L)	"
509	Miss M.McG	18	23269	T.B. Pyosalp. (R)	"
510	Mrs. E.C.	38	23284	Simple cyst (L)	"
511	" F.S.	25	23282	Foll. cysts (F)	"
512	" M.McA	63	23282	Pseudomuc.cyst (L)	"
513	" M.W.	43	7420	"	"
514	" J.P.	41	14857	Dermoid (R)	"
515	" J.M.	29	23567	Gran.cell tumour (L)	"
516	" J.W.	26	22317	Ov.Preg.	"
517	" M.B.	34	Pr.378	Endomet.	"
518	" M.M.	37	23457	" (L)	"
519	" M.H.	26	28381	Pseudomuc.cyst (R)	"
520	" M.R.	57	Pr.396	" cysts (R & L)	"
521	" M.McP	59	14612	" cyst (R)	"
522	" M.R.	32	7532	Foll.cysts (R & L)	"
523/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
523	Mrs. M.C.	42	23605	Endomet. (R & L)	Nil
524	" M.P.	37	15012	Pseudomuc.cyst (R)	"
				Simple cyst (L)	"
525	" M.M.	31	23615	Lut.cyst (R)	"
526	" M.A.	55	23606	Adenocarcin. ov. (R & L)	"
527	" E.H.	25	7657	Simple cyst (L)	"
528	" J.B.	35	15079	"	"
529	" M.McD	35	7634	Ser.cyst (R)	"
530	" C.C.	57	23697	Simple cyst (R)	Sub.total Hyst. 25 years ago
531	" M.H.	43	7714	Dermoid (L)	Nil
532	" B.D.	29	13603	Corp.lut. (L)	"
533	" M.P.	50	Priv.	Chron. Salp. (R)	"
534	" C.H.	41	7792	Chron.Ooph. (R)	"
535	" W.M.	32	23914	Ectopic Preg. (R)	"
536	" M.B.	48	23921	Pseudomuc. cyst (L)	"
537	" M.McC	59	Pr.446	Fibroma (L)	"
538	" P.D.	29	7785	Corp.lut. (L)	"
539	Miss M.B.	28	7805	Endomet (R)	"
540	" M.C.	61	23975	Cystadenofib. (L)	"
541	Mrs. M.C.	40	24030	Simple cyst (L)	"
542	" M.J.	43	7783	"	"
543	" M.M.	31	Pr.459	Pseudomuc.cyst (R)	"
544	" J.C.	32	23596	Ooph. (R)	"
545	" C.McG	30	15366	Endomet. (L)	" "
546	" A.R.	48	24043	Dysgerm. (R)	"
547	" M.T.	59	24061	Gran.Tumour (R)	"
548	" M.D.	20	24059	Pseudomuc.cyst (R)	"
549	" M.L.	49	24036	Cyst. Carcinoma	"
550	" A.D.	33	24083	Pseudomuc.(L)	"
551	" I.M.	68	7891	Ser.cysta.(L)	"
552	" C.B.	26	7933	Pseudomuc.cyst (L)	"
553	" M.C.	44	7983	Carcinoma of ov. (R & L)	"
554	" A.P.	28	8006	Ser. cyst (R)	"
555	" M.K.	23	24261	Simple cyst (L)	"
556	" M.C.	29	24257	Pseudomuc. cyst (L)	"
557	" M.S.	53	24342	Ser.cyst (R & L)	"
558	" C.McA	39	24313	" (L)	"
559	" J.W.	25	25566	Ectopic (L)	"
560	" K.L.	30	15599	Ser.cyst (L)	"
561	Miss I.H.	29	15575	"	"
562	Mrs. I.C.	45	8045	Ser.cyst (L)	"
563	" A.R.	47	1575	" (R)	"
564	" J.W.	32	6143	Endomet. (R)	"
565	" M.R.	31	24475	Corp.lut. (R)	"
566	" E.S.	25	8147	Pseudomuc. cyst. (R)	"
567/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
567	Mrs. M.McP	38	3583	Simple cyst (L)	Nil
568	" J.H.	45	15711	Ser.cyst	"
569	" J.B.	48	23769	Simple cyst (L)	"
570	Miss M.D.	52	12544	Carcinoma of Ov.	"
571	Mrs. R.B.	25	24559	Salp.ooph. (R & L)	"
572	" A.M.	51	22021	Pseudomuc. (L)	"
573	" M.H.	35	24719	Do.	"

1952

574	Mrs. M.J.	57	15852	Cystad. (R)	"
575	" E.H.	56	15880	Pseudomuc. (L)	"
576	" M.J.	34	24580	Ectopic Preg. (R)	"
577	" M.H.	29	24312	Ser.cyst (L)	"
578	" S.B.	68	8322	Pseudomuc.cyst (L)	"
579	" O.B.	30	15888	Ser.cyst (L)	"
580	" C.F.	25	2480	Corp.lut. (R)	"
581	" A.F.	25	14682	Fibroma (R)	"
582	" M.McM	50	24889	Carcinoma of ov. (R & L)	"
583	" A.McT	40	15951	Ser.cyst (R)	"
584	" M.McN	46	4095	Krukenberg (R)	"
585	" B.McE	57	24908	Simple cysts (R & L)	"
586	" A.W.	22	15980	Foll. Cyst (R)	"
587	" M.H.	33	1370	Endomet. (R & L)	"
588	" A.E.	26	1384	Ser.cyst (R)	"
589	" M.F.	21	8432	Pseudomuc. cyst (L)	"
590	" K.M.	55	2465	Demoid (L)	"
591	" I.W.	55	8464	Pseudomuc. (L)	"
592	" M.McD	36	25028	Corp.lut. (R)	"
593	" J.C.	22	25038	Foll.cysts (L)	"
594	" A.P.	37	8324	Simple cyst (R)	"
595	" I.L.	27	1381	Foll.cyst (L)	"
596	" M.H.	23	25052	Corp.lut. (L)	"
597	Miss M.R.	66	13410	Thecoma (R)	"
				Fibroma (L)	"
598	" M.H.	50	25084	Chron.salp.ooph. (R & L)	"
599	Mrs. A.S.	35	25160	Foll.cyst (L)	"
600	" C.R.	31	15105	Ser.cysts (R)	"
601	" N.McC	19	25146	Corp.lut. (L)	"
602	" S.F.	34	16135	Foll.cyst (L)	"
603	" E.R.	43	25289	Corp.lut. (R)	"
604	" A.S.	60	16198	Ser.cyst (L)	"
605	" M.C.	29	8580	Endomet. (L)	"
606	" E.R.	48	10894	Pseudomuc.cyst (L)	"
607	" E.F.	30	8128	Salp.ooph. (L)	"
608	" M.B.	37	4840	Corp.lut. (L)	"
609/					



<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
652	Mrs. M.C.	36	1221B	Ectopic Preg. (L)	Nil
653	" M.G.	62	26007	Brenner (R)	"
654	" N.P.	36	17034	Pseudomuc. cyst	"
655	" E.M.	21	25767	Ser.cyst (L)	"
656	" R.S.	72	3317	Pseudomuc. cyst (R)	"
657	" A.M.	54	225	Fibroma (R)	"
				Oophoritis (L)	"
658	" E.D.	45	26059	Corp.lut. (R)	"
659	" J.McF	36	26060	Uniloc.cyst (L)	"
660	" E.W.	33	6771	Corp.lut. (R)	"
661	" I.W.	58	26009	Adenocarcinoma ov. (R)	"
662	" C.G.	33	26090	Ser.cyst (L)	"
663	" C.McK	29	26071	"	"
664	" M.McK	46	9139	Fibroma	"
665	" J.G.	37	23655	No report	"
666	" M.L.	47	17100	Ser.cystad.	"
667	" C.F.	49	267 Pr.	Simple cyst (L)	"
668	" W.C.	26	14889	Ser.cysts (R & L)	"
669	" J.B.	25	26184	Foll.cysts (L)	"
670	" R.H.	35	271 Pr.	Corp.lut. (R)	"
671	" M.D.	35	23661	Blood cysts (L)	"
672	" E.L.	58	26205	Ser.cyst (R & L)	"
673	" C.McA	38	26236	Fibroma (L)	"
674	" M.J.	69	17230	Pseudomuc.cyst (R)	"
675	" M.S.	47	26289	Ser.cyst (R)	"
676	" M.S.	34	26314	Hydrosalp. (L)	"
677	Miss I.S.	24	17322	Fibroma (L)	"
678	Mrs. C.L.	33	9289	Pseudomuc.cyst	"
679	" M.G.	23	9285	Fibroma	"
680	" A.M.	53	Priv.	Ser.cyst (R)	"
681	" E.D.	50	17368	Pseudomuc.cyst (R)	"
682	" A.C.	57	9329	Dermoid (L)	"
683	" E.D.	36	17360	" (R)	"
684	" M.J.	34	9507	Foll.cyst (R)	"
685	" M.McL	27	17413	Ectopic (L)	"
686	" M.G.	30	20161	Corp.lut. (L)	"
687	" M.B.	47	9367	Pseudomuc.cyst (L)	"
688	" M.E.	41	17440	"	"
				Cystadenocarcinoma (L)	"
689	" S.McN	33	26549	Chron.salp.ooph. (L)	"
690	Miss C.J.	44	17438	Hydrosalp. (R & L)	"
691	Mrs. E.S.	32	25853	Lut.cyst. (R)	"
692	Miss C.B.	16	7181	Ser.cyst (L)	"
693	" M.T.	19	Pr.	Tubo-ov.abcess (L)	"
694	Mrs. A.C.	74	16395	Pseudomuc. cyst (L)	"
695	" W.D.	26	353 Pr.	Endomet. (L)	"
696/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
609	Mrs. M.B.	38	16500	Corp.lut. (L)	Nil
610	" E.McA	52	8651	Simple cyst (R)	"
611	" M.M.	70	8672	Ser.cyst (L)	"
612	" H.D.	45	16279	" (R)	"
613	" E.L.	35	18870	Corp.lut.(R)	"
614	Miss M.B.	43	8725	Cystaden.carcinoma (L)	"
615	Mrs. D.A.	29	20529	Ser.cyst (R)	"
616	Miss I.E.	49	14713	Pseudomuc.cyst (R)	"
617	Mrs. M.W.	53	25508	Simple cyst (L)	"
618	" M.M.	36	8805	Ser.cyst (L)	"
619	" J.M.	30	13156	Tubo-ov. abcess (L)	"
620	Miss E.H.	34	25537	Ectopic	"
621	Mrs. P.R.	50	25540	Fibroma (R & L)	"
622	" M.P.	57	16353	Pseudomuc. cyst (L)	"
623	" M.S.	32	25519	Ser.cyst (R)	"
624	" M.H.	44	Priv.	Pseudomuc.cysts (R & L)	"
625	" M.L.	23	8816	Ectopic Preg. (R)	"
626	" M.G.	37	16524	Simple cyst (L)	"
627	" A.F.	41	25600	Pyosalpinx (L)	"
628	" I.C.	41	20561	Foll.cyst (R & L)	"
629	" S.M.	35	13397	Ectopic Preg. (L)	"
630	" J.M.	32	16390	Foll.cyst (L)	"
631	" V.T.	52	256016	Dermoid (L)	"
632	" M.M.	32	16384	Endomet. (R)	"
633	" M.C.	33	169B	Foll.cyst (L)	"
634	Miss A.R.	17	16422	Ser.cyst. (L)	"
635	Mrs. M.K.	29	16349	Ectopic preg. (L)	"
636	" M.A.	36	Priv.	Adeno.carcinoma ov. (L)	"
637	" A.A.	71	16477	Ser.cyst (L)	"
638	" C.McG	32	15366	Foll.cyst (R)	"
639	" M.W.	40	25756	Salp.ooph. (R)	"
640	Miss A.I.	34	16505	"	"
641	Mrs. A.C.	27	15619	Ectopic Preg. (R & L)	"
642	Miss E.P.	18	8957	Ser.cyst (R)	"
643	Mrs. M.B.	47	16565	Choc.cysts (R & L)	"
644	" M.McK	37	Pr.	" (R)	"
645	" M.S.	30	25908	Ser.cyst (R)	"
646	" A.B.	35	25588	"Blood cyst" (L)	"
647	" A.McD	36	25957	Ser.cyst (R)	"
648	" E.C.	25	6279	Corp.lut. (R)	"
649	" M.B.	50	23618	Gran.cell (L)	"

1953

650	" J.H.	31	25203	Endomet. (L)	"
651	" R.H.	45	25982	Pseudomuc. cyst	"
652/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
696	Mrs. M. McK	33	26686	Foll. cyst (R)	Nil
697	" M. McA	62	26734	Pseudomuc. cyst (R)	"
698	" A.H.	25	17551	" (L)	"
699	" A.B.	37	16478	" (R)	"
700	" J.M.	81	17650	Fibroma (R)	"
701	" L.S.	69	9573	Adenocarcinoma ov. (R)	"
702	Miss C.G.	20	9580	Pseudomuc. cyst (R)	"
703	Mrs. A.T.	68	26880	"	"
704	" J.H.	33	26908	Corp. lut. (R)	"
705	" M.W.	33	26917	Pseudomuc. cyst (R)	"
706	" F.B.	55	16187	" (L)	"
707	" M.S.	37	17759	Uniloc. cyst (L)	"
708	" J. McL	62	26998	Ser. cystad. (R)	"
709	" J.W.	39	27013	Endomet. (L)	"
710	" A.C.	44	Priv.	Dermoids (R & L)	"
711	" E.B.	26	27058	Pseudomuc. (L)	"
712	" E. McC	63	9683	Adenocarcinoma ov. (R & L)	"
713	" J.T.	53	27029	Pseudomuc. cystad. carcinoma (R)	"
714	" J.C.	38	9746	Dermoid (L)	"

1954

715	" J.R.	34	9797	Foll. cyst (R & L)	"
716	" I. McK	23	27133	T.B. Salp. ooph. (R)	"
717	" I.T.	26	17391	Foll. cyst (R & L)	"
718	" E.M.	33	27237	" (L)	"
719	" M.S.	37	17926	Dermoid (L)	"
720	" A. McK	28	18080	Ectopic (R)	"
721	" M.C.	45	Priv.	Ovar. Haem. (R)	"
722	" O. McG	35	18035	"	"
723	Miss J.L.	20	18071	Simple cyst (R)	"
724	" S.W.	34	18170	Tubo-ov. abscess (L)	"
725	Mrs. E.L.	48	18237	Pseudomuc. cyst (R & L)	"
726	" A.B.	24	18233	" (R)	"
727	" L.P.	34	10066	Dermoid (L)	"
728	" M. McG	78	10058	Pseudomuc. cyst (L)	"
729	" S.G.	29	10080	Simple cyst (L)	"
730	" D. McK	45	18255	Fibroma (R & L)	"
731	" I.H.	52	27703	Ser. cyst (L)	"
732	" E. McC	53	10111	Ser. cyst (R)	"
733	" J.D.	60	10108	Pseudomuc. cyst (L)	"
734	" M.H.	22	10121	Foll. cyst (L)	"
735	" M.R.	28	from	Ser. cystad. (L)	"

Mat. Hos.

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
736	Mrs. H.S.	36	18325	Pseudomuc. cyst (L)	Nil
737	" A.McB	33	15880	Salpingitis (L)	"
738	Miss M.B.	15	18377	Dermoid (L)	"
739	Mrs. A.W.	60	18309	Ser.cystad. (L)	"
740	" A.G.	33	10184	Ectopic (L)	"
741	" H.E.	25	29907	Rt.salp.ooph.	"
742	" M.N.	19	10217	Pseudomuc. cyst (L)	"
743	" M.J.	43	27907	Simple cyst (R)	"
744	" A.M.	26	10226	Ser.cystad. (L)	"
745	" E.Y.	27	12934	"	"
746	" M.S.	32	10273	Tubal.preg.	"
747	" S.D.	64	18483	Ser.cyst (R)	"
748	" I.A.	43	28079	Pseudomuc cyst (R)	"
749	" E.A.	53	9142	Fibroma (R)	"
750	" H.P.	21	28158	"	"
751	" M.R.	21	18543	" (L)	"
752	" B.D.	32	10370	Subacute salp. (L)	"
753	" M.F.	30	10365	Pseudomuc. cyst (R)	"
754	" C.M.	32	27917	Ser.cyst (R)	"
755	" B.L.	40	28163	Fibroma (R)	"
756	" H.McR	33	28256	Ser.cystad. (R)	"
757	" M.S.	31	27169	Corp.lut. (R)	"
758	" R.R.	36	2829	Dermoid (L)	"
759	" I.L.	30	17885	Endomet. (R)	"
760	" J.M.	39	29306	Corp.lut (L)	"
761	Miss L.McC	57	28384	Ser.cyst (R)	"
762	Mrs. C.S.	28	10484	" (L)	"
763	Miss F.McD	27	10491	Ser.cyst (L)	"
764	Mrs. M.S.	44	18709	Adrenal tum. of ov. (L)	"
765	" M.M.	24	10511	Foll.cyst (L)	"
766	Miss M.C.	23	Pr.	Ser.cyst (R & L)	"
767	" E.R.	21	18826	Pseudomuc.cyst (L)	"

1955

768	Mrs. C.McK	37	10560	Corp.lut. (L)	"
769	" G.F.	31	24840	Ser.cyst (R)	"
770	" A.D.	61	28266	Pseudomuc. cyst (R)	"
771	" A.B.	21	18774	Ser.cyst (R)	"
772	" M.O'R	25	10620	Pseudomuc. cyst (L)	"
773	" E.McL	21	10660	Lut.cyst (L)	"
774	" W.W.	52	55B Pr.	Adenocarcinoma of ov. (origin unknown)	Prev.Hyster. & removal of Rt. Ovary
775	" M.C.	34	18963	Ectopic (L)	Nil
776/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
776	Mrs. M.C.	34	18963	Ectopic (L)	Nil
777	" M.J.	49	3498	Fibroma (R & L)	"
778	" M.A.	79	28584	Ser.cyst (E & L)	"
779	" R.M.	41	11468	Ser.cyst (L)	"
780	" M.McG	25	12054	Ser.cyst	"
781	" S.McA	27	28757	Foll.cyst	"
782	" A.B.	35	28725	Ser.cyst	"
783	Miss F.F.	26	12065	Salp.ooph. (R)	"
784	Mrs. E.G.	38	28519	Ser.cyst (R)	"
785	" J.W.	27	28505	T.B. Salp.ooph.(L)	"
786	" M.A.	51	Pr.	Carcinoma of Ov.	"
787	" M.K.	32	24097	Fibroma (R)	"
788	" S.S.	47	11154	Ser.cyst (R & L)	"
789	" H.H.	34	28700	Corp.lut. (R)	"
790	" M.W.	51	28837	Ser.cyst (R)	"
791	" M.T.	60	Priv.	Ser.cystad.carcinoma	"
792	Miss M.F.	46	10870	Abcess of Corp.lut.	"
793	Mrs. G.S.	50	28842	Pseudomuc. cyst	"
794	" M.F.	44	9927	Chron.salp.ooph.(R)	"
795	" A.F.	30	10899	Pseudomuc. cyst (R)	"
796	" M.McN	28	29014	Pseudomuc. cyst (L)	"
797	" E.A.	38	10945	Ser.cyst (L)	"
798	" M.M.	29	6979	Foll.cyst (R)	"
799	" A.H.	27	11005	Ser.cyst (R)	"
800	" M.McL	25	28387	"	"
801	" E.B.	28	19209	Dermoid	"
802	" C.K.	28	11029	Pseudomuc.cyst	"
803	Miss I.B.	18	28805	Foll.cyst	"
804	Mrs. I.T.	37	28199	Ser.cyst (R)	"
805	" G.McE	27	7860	Dermoid (R)	"
806	" M.C.	41	11051	Pseudomuc. cyst (R)	"
807	" J.A.	72	19254	"	"
808	" E.McA	25	11090	Corp.lut.haema.	"
809	" H.J.	27	29109	T.B. Salp.	"
810	" E.F.	63	29238	Ser.cyst (R)	"
811	" E.R.	33	11076	Chron.Salp.ooph.(L)	"
812	" C.D.	28	28692	Ectopic Preg. (R)	"
813	" M.J.	29	8568	Corp.lut. (R)	"
814	" A.R.	56	13059	Ser.cyst (R)	"
815	" M.McG	43	Priv.	Endomet. (R & L)	"
816	" G.A.	37	8278	Pseudomuc.cyst	"
817	" P.T.	30	29393	Foll.cyst (L)	"
818	" D.S.	56	19360	Papil.adeno-carcinoma (L)	"
819	" C.C.	35	19369	Corp.lut. haema.	"
820	" M.M.	37	26406	Hydrosalp.(L)	"
821/					

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
821	Mrs. A.C.	33	29492	No report	Nil
822	" G.G.	64	29487	Pseudomuc.Cyst (L)	"
823	" N.L.	32	19454	Ser.Cyst (L)	"
824	" M.McC	32	11291	Simple cyst (L)	"
825	Miss I.S.	27	19431	Pseudomuc.Cyst (L)	"
826	Mrs. M.C.	28	19452	Do.	"
827	" M.L.	41	29559	Tubo-ov. T.B. (L)	"

Cases of Laparotomy in which the pathology was known  
to be ovarian or possibly ovarian

1930

828	Mrs. C.McI	36	1615	Salp.oophoritis	"
829	" A.S.	31	1618	Chron. Salp.oophoritis	"

1931

830	" N.C.	29	1933	Chron. Salp.oophoritis	"
831	" M.McF	46	1963	Carcinomatosis	"

1933

832	" S.A.	59	2661	Carcinoma of unknown origin	"
833	" M.McK	50	3191	Carcinomatosis	"
834	" M.McL	60	3207	Carcinoma of Ov.	"
835	" M.F.	39	3346	Sarcoma of Ov.	"

1934

836	" I.S.	28	3385	Chron.Salp.oophoritis	"
837	" M.N.	66	1027	Pelv.carcinoma.unknown origin	"

1935

838	Miss E.L.	49	5401	Pelv.malig.dis. origin unknown	"
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<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
<u>1936</u>					
839	Mrs. G.M.	29	6585	Chron.Salp.oophoritis	Nil
840	" A.F.	62	6659	Carcinomatosis. origin unknown	"
841	" M.McG	55	6689	Carcinoma of Ov.	"
842	Miss V.S.	24	6793	Sarcoma of Ov.	"
843	" I.W.	21	6920	T.B. Salp.oophoritis	"
844	Mrs. C.W.	26	6981	Do.	"
<u>1939</u>					
845	" J.McI	39	2958	Chron.Salp.oophoritis	"
846	" M.M.		10379	Carcinoma of Ov.	"
<u>1940</u>					
847	" L.A.		11227	Carcinoma of Ov.	"
848	" F.J.	30	11254	Do.	"
<u>1941</u>					
849	" M.O'B	33	11772	Carcinoma of Ov.	"
850	" A.C.	30	12704	Do. of Ovaries	"
<u>1942</u>					
851	" A.P.	41	13334	Carcinoma of Ov.	"
852	" A.T.	53	13493	Do.	"
853	" A.C.	25	13614	T.B. Salp. Oophoritis	"
854	" H.B.	41	13493	Carcinoma of Ov.	"
<u>1944</u>					
855	" C.R.	47	15464	Carcinoma of Ovaries	"
856	" S.O'B	42	15544	Do. of Ov.	"
<u>1945</u>					
857	" J.C.	35	17084	Carcinoma of Ovaries	"
<u>1946</u>					
858	" M.McL	45	17902	Carcinoma of Ovary	"
859	" M.L.	52	18049	Carcinoma of Rt.Ov.	"

<u>No.</u>	<u>Initials</u>	<u>Age</u>	<u>Clinic Number</u>	<u>Pathology</u>	<u>Previous Hysterectomy</u>
<u>1947</u>					
860	Mrs. E.M.	26	16062	Carcinoma of Ov.	Nil
<u>1948</u>					
861	" M.C.	52	15094	Carcinoma of Ov.	"
862	" M.H.	62	62B	Carcinoma of unknown origin	"
<u>1949</u>					
863	" M.P.	38	15361	Secondary carcinomatosis nodule	"
<u>1952</u>					
864	" A.G.	48	24694	Adenocarcinoma of Ov.	"
865	" A.C.	54	B.52	Carcinomatosis of Peritoneum	"
866	" I.G.	47	16421	Carcinoma of Ovaries	"
867	" E.R.	46	16518	Adenocarcinoma of Ov.	"
868	" M.G.	58	23710	Adenocarcinoma	"
869	" M.C.	71		Do. unknown origin	"
<u>1954</u>					
870	" R.A.	54	18288	Adenocarcinoma of unknown origin	"
871	" D.M.	45	18593	Sarcoma of unknown origin	"
<u>1955</u>					
872	" I.F.	75	11049	Adenocarcinoma of unknown origin	"



O V A R I A N   A C T I V I T Y   F O L L O W I N G

H Y S T E R E C T O M Y

(An inquiry into the character, degree and duration of ovarian activity following hysterectomy, with an additional section on the incidence of pathological change in ovaries that are conserved when the uterus is removed)

by

ROBERT G. WHITELOW, M.A., M.B.Ch.B., M.R.C.O.G.

P R E F A C E

Although the idea of investigating the problem of ovarian activity following hysterectomy by the various methods described in this thesis was my own conception, I wish to express my sincere thanks to a number of people whose kindness and specialised knowledge have enabled me to achieve this object.

First of all I wish to thank Dr. L.H. Easson, Biochemist of the Fife District Laboratories, who supervised the performance of the pregnanediol estimations and also Miss Barbara Wilson B.Sc., who performed them on my behalf, often at times more suited to the requirements of my tests than her own convenience.

I also acknowledge my indebtedness to Miss Rhona McBride of the Department of Obstetrics and Gynaecology of the University of Edinburgh, who instructed me in the collection and staining of vaginal smears, as well as to Mr. Rutherford of the Fife District Laboratories who advised me on staining technique.

My ignorance of German obliged me to seek the assistance of translators when I wished to peruse medical articles written in that language, and in this respect I was well served/

served by Mrs. Edith Duncan, Dr. Gruneberg and my wife.

These also deserve my thanks.

As the performance of bio-assays such as those described in the fifth investigation, could not, with any degree of accuracy, have been undertaken by me, since I have no experience of the techniques of animal assays, I consider myself fortunate to have had Dr. B. M. Hobson of the Usher Institute undertake these estimations at my request, and gratefully acknowledge his valuable assistance.

Much of the cost of these investigations was met by a financial grant which I received from the Endowments Fund of the West Fife Group of Hospitals. To the Board of Management who voted me this grant, and to the Advisory Committee on Medical Research for Scotland who recommended that it be made available to me, I am also deeply grateful.

Lastly, I should like to thank Professor R. J. Keller of the University of Edinburgh for his encouragement and helpful advice, Dr. T. A. Gillie, Gynaecologist of the Dunfermline and West Fife Hospital for access to his case records, and the women who were the subjects of the various investigations for their patient and loyal co-operation.

R. G. WHITELOW

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PART I

OVARIAN ACTIVITY FOLLOWING HYSTERECTOMY



THE NEW ZEALAND

epidemiological systems may be said to have been, upon performing the operation a year or two of their routine simultaneous studies, all under the microscope, at least in a general sense, as to the general nature of the general principles of their design for the purpose of the general nature of the

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GENERAL INTRODUCTION

epidemiological systems may be said to have been, upon performing the operation a year or two of their routine simultaneous studies, all under the microscope, at least in a general sense, as to the general principles of their design for the purpose of the general nature of the

These words being it is to describe possible usually attempts to justify their attitude by placing a natural reluctance

THE TWO VIEWPOINTS

Gynaecological surgeons may be said to be divisible into those who, when performing the operation of hysterectomy, make it part of their routine simultaneously to remove the ovaries, and those who endeavour, at least in the pre-menopausal patient, to conserve all possible ovarian tissue, unless gonadal pathology or pelvic malignant disease dictates for them a more radical course of action.

Protagonists of bilateral oophorectomy offer two arguments in support of their practice. The first is that once the uterus has been removed ovarian atrophy and cessation of function soon follow, and the second is that removal of a woman's ovaries precludes the possibility of that particular patient developing, at a subsequent date, carcinoma of these organs.

Those whose policy it is to conserve the ovaries whenever possible usually attempt to justify their more cautious attitude by pleading a natural reluctance to excise tissue which has the appearance of being healthy and still functioning, adding perhaps a word of scepticism concerning the/

the alleged early atrophy of the ovaries which is said by some to occur after hysterectomy has been performed. They sometimes give, as an additional reason for conservatism, their belief that oophorectomy, when carried out on a woman during the reproductive period of life, by abruptly depriving her of the ovarian hormones, is likely to precipitate a more severe menopause than that which occurs as a consequence of the gradual physiological decline in ovarian activity which, they contend, ensues if the ovaries are left at operation.

Typical of those who hold that removal of the uterus ought also to imply the simultaneous removal of both ovaries was the late Professor James Hendry who writes as follows:-

"When the uterus has been removed, the balance of clinical evidence is that the conservation of ovarian tissue, even under the most favourable conditions, makes little difference to the onset of menopausal symptoms. The variation in individual cases is hardly greater than the menopausal variations in healthy women." (Hendry 1936).

It is unfortunate, however, that perusal of the article from which the above quotation has been taken, reveals remarkably little evidence which either supports or refutes its author's contention. Professor Hendry refers to the results of the investigation into the menopause conducted by/

by the Council of the Medical Women's Federation (1933). This disclosed that, of the thousand women interrogated, 62.3% suffered from hot flushes and "10.3% were definitely "incapacitated", whereas only "15.8% passed through the "menopause without symptoms". From these figures, Professor Hendry seems to imply that any discomfort that may attend a surgical castration is not likely to be much worse than that of a physiological menopause. No comparison, however, is made between the symptomatology of those whose ovaries have been removed and those whose ovaries have been conserved, and the value of this article as a contribution to the elucidation of the problem on which it attempts to advise, is minimised by a total absence of statistical detail concerning the oophorectomised subject.

Moreover, Sessums and Murphy (1932) in an analysis of 91 cases of hysterectomy with complete or partial conservation of the ovaries in women all of whom were under the age of 36 at the time of operation, have reported the appearance of what they considered to be unmistakable symptoms of the menopause in 43.9% of their subjects before they had reached the age of 40, a finding which would tend to/

to suggest that hysterectomy, even when performed with conservation of the ovaries, may sometimes result in a premature decline in ovarian activity.

On the other hand, Richards (1951) in his survey of vasomotor phenomena following hysterectomy, discovered that the incidence of hot flushes diminished as the amount of conserved ovarian tissue increased. He found hot flushes to be most prevalent in subjects who had been deprived of both ovaries, much less frequent in those who had lost one ovary and least common where both ovaries had been retained in situ.

Burford and Diddle, (1936) in a detailed review of the literature relating to the ovarian changes which are said to have occurred following hysterectomy, are able to quote a number of writers, mainly German, who, in spite of minor discrepancies, seem to concur in the belief that removal of the human uterus hastens ovarian atrophy and an apparent cessation of ovulation. Werth, for example, is reported by these writers, as having observed

"an increase in the size of the follicles with  
"conspicuous absence of follicular ova in human ovaries  
"following hysterectomy",

while Keitler is said to have noted what he thought was

"a/

"a significant diminution in the number of  
"follicles after excision of the uterus".

A similar observation is attributed to Jacobsohn. Then, Vineberg and Hawks working independently are stated to have found cystic ovaries when performing laparotomies upon women who had already undergone the operation of hysterectomy. Lindig is mentioned as having seen enlargement of the follicles after removal of the uterus, Terada as having observed follicular atresia as a sequel to the same operation, and Schubert as being unable to find ova after uterine excision.

Formidable though this list of reports appears, it does not prove that all ovarian activity ceases after hysterectomy, though it presents evidence that, in certain instances, removal of the uterus would appear to have been followed by a cessation of ovulation.

It is not difficult, however, to quote other workers whose observations made at laparotomies performed years after hysterectomy, are completely different from those mentioned in this catalogue of ovarian decline and failure. Of these Victor Bonney may be regarded as an example. Concerning/

Concerning the belief that ovaries conserved at hysterectomy soon exhibit atrophic or degenerative change, he makes this remark:-

"I have had many opportunities of seeing ovaries "through an abdominal incision at various periods after "hysterectomy and they appear perfectly normal".  
(Bonney 1937).

While a normal macroscopic appearance is no guarantee of unimpaired function, it is equally true that naked eye impressions of ovarian degeneration such as have been mentioned need not necessarily denote that ovarian activity has come to an end.

Bonney also adds a necessary note of warning to those who, on the basis of what appear to be menopausal symptoms, argue that hysterectomy has produced a deterioration in ovarian activity, by stating:-

"It is forgotten that most hysterectomies are "carried out on patients over forty whose normal "climacteric is not more than eight or ten years distant".

He is equally emphatic on the subject of the occurrence of ovarian pathology in conserved ovaries, on which he writes as follows:-

"The number of times I have had to operate on "ovaries conserved at a hysterectomy can be counted on "the fingers of one hand and my experience is long and "large".

Though/

Though this is hardly a conclusive argument, it is a significant comment to come from one who is said to have performed thousands of hysterectomies.

Those whose policy and habit it is to spare the ovaries at hysterectomy can produce evidence that the menopause of castration is a severe one in comparison with a physiological menopause.

Using the vaginal smear as an index of the endocrine function of the ovary, Papanicolaou (1936) has shown that the atrophic changes which occur in the vaginal epithelium following the menopause are more pronounced when this is the result of bilateral oophorectomy, and Bishop (1950) referring to its symptomatic manifestations states that the artificial menopause

"with its sudden deprivation of oestrogens is  
"nearly always stormy".

It comes indeed as no surprise to discover that the American S. R. N. Reynolds, in his book "The Physiology of the Uterus", devotes eight pages to the endocrinological effects of hysterectomy and that this section of the work, which contains eighty-three references, is summarised by the author as follows:-

"The/



"The effects of hysterectomy upon the ovary are  
 "today the subject of divided opinion despite an  
 "abundance of clinical and experimental consideration  
 "by eminent clinicians and investigators for more than  
 "sixty years". (Reynolds 1949).

It would appear, therefore, that neither those who remove,  
 nor those who conserve the ovaries at hysterectomy do so  
 from arguments that rest upon an unassailable foundation  
 of established scientific fact.

That this should still be so is the reason why the  
 present investigation has been undertaken, for the subject  
 is not only of intrinsic scientific interest but also of  
 considerable clinical importance.

GENERAL PLAN OF INVESTIGATION

Although the character, degree and duration of ovarian activity following hysterectomy are subjects which have aroused the curiosity of many workers, only a handful of the many investigators have attempted to tackle the problem from more than one aspect, and most of the published work is weakened by a total neglect of controls.

It is clear, however, that if use were to be made of a variety of methods of assessing ovarian activity and these were to produce a general measure of agreement, the conclusions could then be considered to have a greater significance, since the fortuitous effects of non-gonadal influences would thus be minimised, and errors due to adventitious factors would tend to cancel each other in the results.

It is also obvious that if reliable conclusions are to be drawn about the activity of conserved ovaries, these ought to be reached after the results obtained from hysterectomised subjects whose ovaries have been conserved are compared with those obtained from women from whom both uterus and ovaries have/

have been removed, and also with those obtained from women from whom neither uterus nor ovaries have been excised.

For this reason it was decided that in this piece of research three groups of subject would be investigated.

These are:-

- (1) Group (a) consisting of women who had undergone, prior to the menopause, the operation of hysterectomy with conservation of one or both ovaries,
- (2) Group (b) consisting of women of comparable age group and pelvic pathology who had undergone, prior to the menopause, the operation of hysterectomy with bilateral salpingo-oophorectomy, and
- (3) Group (c) consisting of women of comparable age group, who had undergone neither operation but whose uteri and ovaries were still in situ and apparently functioning.

To obtain the subjects for groups (a) and (b) letters were sent to ex-patients of the Dunfermline and West Fife Hospital, who had, at least one year previously, undergone the operation of hysterectomy, inviting them to assist in this research project.

About ninety women were initially persuaded to participate, but with the passage of time, these numbers dwindled. Some, for a variety of reasons, found that they could not continue to co-operate; others were unable or unwilling to keep reliable and regular temperature records; a few left the district before the work was completed. The result was that/

that, at the end of eighteen months, there remained

- (1) twenty-four women who had undergone the operation of hysterectomy with conservation of one or both ovaries, and
- (2) twenty-four women who had undergone the operation of hysterectomy with bilateral salpingo-oophorectomy.

To obtain the support of women who would prove suitable subjects for group (c) was more difficult.

To overcome this difficulty the records of the Dunfermline Maternity Hospital were examined, and from these were obtained the names of fourteen women of ages approximately comparable to those of groups (a) and (b), each of whom resided within a radius of about one mile of the hospital. This proximity and the fact that all these women had, within the past two years, been confined in the Dunfermline Maternity Hospital, enabled one to enlist their co-operation.

It would have been preferable to have had twenty-four subjects belonging to this group, but since they represent what, for want of a better term one may call the "normal woman", the results they returned, when subjected to the various tests, were, to a great extent, predictable, this type of woman having frequently been the subject of detailed and accurate study/

study both by gynaecologists and endocrinologists.

It was initially thought that all three groups of women should be investigated from five different aspects and it was also hoped that every woman in each group would be individually investigated by each of the five different methods. This aim was achieved for the first four investigations, but for reasons which will be mentioned later, it was not considered necessary to apply the fifth method of investigation to each subject of all three groups.

The problem of ovarian activity following hysterectomy was investigated by the following methods:-

- (1) By the menopausal index, i.e. by a mathematical assessment of the severity of the menopausal syndrome made on the basis of a points system.
- (2) By the use of temperature records which, if they exhibited the biphasic pattern characteristic of the ovulating subject, might be considered as evidence of the persistence of ovulation following hysterectomy.
- (3) By the use of urinary pregnanediol estimations designed to check the accuracy of the conclusions reached by means of the temperature records.
- (4) By the use of vaginal smears stained to show evidence of cornification and therefore employed as an index of the oestrogenic activity of the conserved ovaries.
- (5) By bio-assay of pituitary gonadotrophin. The argument underlying this method is that when ovarian oestrogenic activity/

activity subsides following the menopause, pituitary gonadotrophin is released from the inhibitory influence which ovarian oestrogen is believed to exercise upon it, and its concentration in the blood and urine of the subject is considerably increased. By performing bio-assays of this hormone with the urine of the subject being investigated, one therefore indirectly forms an estimate of the strength of the inhibitory influence of the ovarian oestrogen and consequently of the functional state of the ovaries.

The object of these investigations was to attempt to find an answer to the question of whether or not ovarian activity continues after hysterectomy. The results might therefore be said to have some direct bearing on a practical problem which confronts the gynaecological surgeon when performing the operation of hysterectomy for some non-malignant condition. That problem, of course, is whether or not to remove the ovaries.

The chance of a conserved ovary subsequently becoming diseased or even undergoing neoplastic change must also, however, influence the surgeon as he weighs the "pros" and "cons" of oophorectomy.

For this reason it was decided that, in addition to those investigations, an attempt should be made to express in mathematical terms the relative likelihood of this contingency.

The request was made to peruse the records of a large teaching/

teaching hospital and from the evidence there disclosed to make an assessment of the degree of risk to which a patient is subjected when her uterus is removed for some innocent pathological condition, while her ovaries are conserved.

The necessary permission was granted, and it is with such a survey that this thesis concludes.

Special Note on Drugs and Medicines

The women who agreed to participate in this piece of research were asked to refrain from taking any form of drug or medicine during, and for at least two months prior to, the actual performance of the various tests.

These precautions, though applying in particular to synthetic oestrogens, were extended as far as possible to cover all forms of medicine, lest the action of some such agent should interfere with the accuracy of the results.



THE SUBJECTS of the INVESTIGATIONSGroup (a) - Table 1Cases of Hysterectomy with Conservation of Both Ovaries

<u>No.</u>	<u>Initials</u>	<u>Age at time of Opera- tion</u>	<u>Type of Opera- tion (Total or Sub total Hyst.)</u>	<u>Pathology</u>
1a	Mrs. B.D.	37 6/12ths	Total	Caes.Hysterectomy (Accd.Haem.)
2a	" M.M.	41 3/12ths	"	Functional Uterine haemorrhage
3a	" J.F.(D)	46 11/12ths	"	Metropathia Haemorrhagia
4a	" E.D.	45 3/12ths	"	Do.
5a	" J.F.(B)	44 4/12ths	"	Fibroids
6a	" J.C.(K)	39 11/12ths	"	Do.
7a	" M.McD.	40 6/12ths	Subtotal	Do.
8a	" J.C.(B)	34 2/12ths	Total	Functional Uterine Haemorrhage
9a	" M.H.(C)	34 11/12ths	"	Myohyperplasia
10a	" H.T.	38 9/12ths	"	Fibroids
11a	" C.K.	37 5/12ths	Subtotal	Myohyperplasia
12a	" A.R.	44 10/12ths	Total	Metropathia Haemorrhagia
13a	" M.H.(T)	34 7/12ths	Subtotal	Caes.Hysterectomy (Severe Pre- Eclampsia)
14a	" J.S.	37 3/12ths	Total	Caes.Hysterectomy (Accd.Haem.)
15a	" I.R.	43 7/12ths	Subtotal	Do. (Severe hyper- tension)
16a	" J.McI	47	Total	Myohyperplasia

Cases with Conservation of One Ovary

17a	" M.W.	32 1/12th	Subtotal	Fibroids
18a	" H.P.	44 5/12ths	Total	"
19a	" E.C.	44 2/12ths	Subtotal	"
20a	" J.O'N	39	Do.	Caes.Hysterectomy (Mixed Accd. Haemorrhage)
21a/				

<u>No.</u>	<u>Initials</u>	<u>Age at time of Opera- tion</u>	<u>Type of Opera- tion (Total or Sub total total Hyst.)</u>	<u>Pathology</u>
21a	Mrs. M.D.	47 1/12th	Total	Myohyperplasia
22a	" J.B.	35 3/12ths	"	Caes. Hysterectomy (Placenta Praevia. Multiparity)
23a	Miss M.S.	49 2/12ths	"	Fibroids
24a	Mrs. M.C.	34 9/12ths	Subtotal	Fibroids Complicating Pregnancy

All patients were premenopausal at the time of operation.

Average age at time of operation - 40.6 years

Group (b) - Table IICases of Hysterectomy with Removal of Both Ovaries

<u>No.</u>	<u>Initials</u>	<u>Age at time of Opera- tion</u>	<u>Type of Opera- tion (Total or Sub Total Hysterect.</u>	<u>Pathology</u>
1b	Mrs. A.S.	40 5/12ths	Total	Metropathia Haemorrhagia
2b	" A.Y.	42 11/12ths	"	Adenomyosis
3b	" A.C.	43 6/12ths	"	Metropathia Haemorrhagia
4b	" M.W.(C)	30 5/12ths	Subtotal	Abdominal Preg. adherent to Uterus
5b	" H.W.	45 6/12ths	Total	Cystic Glandular Hyperplasia
6b	Miss B.F.	47 8/12ths	"	Fibroids
7b	Mrs. M.B.	40 11/12ths	"	Cystic Glandular Hyperplasia
8b	" J.S.	46 7/12ths	"	Metropathia Haemorrhagia
9b	" M.S.(C)	49 6/12ths	"	Fibroid
10b	" A.H.	38 11/12ths	"	Cystic Glandular Hyperplasia
11b	" M.S.(K)	43 2/12ths	"	Fibroids
12b	" C.O'G	38 10/12ths	"	Myohyperplasia
13b	" M.P.	39 2/12ths	Subtotal	Myohyperplasia
14b	" P.F.	29	Total	Adenomyosis
15b	" A.T.	31 9/12ths	Subtotal	Myohyperplasia
16b	" E.D.	40 4/12ths	Do.	Functional Uterine Haemorrhage
17b	" C.P.	46 9/12ths	Do.	Fibroid
18b	" M.W.(D)	41 11/12ths	Total	"
19b	" E.M.	44	Subtotal	Fibroids
20b	" A.W.	45	Total	Adenomyosis
21b	Miss C.McG	41 7/12ths	"	Pseudomucinous Cyst of Ovary
22b	" A.E.	48 1/12th	Subtotal	Fibroids
23b	Mrs. E.Y.	49 1/12th	Total	Multiple fibroids
24b	" E.H.	45	"	Fibroids

All patients were premenopausal at the time of operation.

Average age at time of operation = 42.06

Group (c) - Table IIIWomen who had undergone neither Hysterectomy nor Oophorectomy

<u>No.</u>	<u>Initials</u>	<u>Age Years</u>
1	Mrs. W.D.	40 1/12th
2	" S.D.	43 4/12ths
3	" M.R.	43
4	" D.R.	38 6/12ths
5	" J.K.	41 5/12ths
6	" J.H.	40
7	" C.W.	48 7/12ths
8	" N.McG	49 2/12ths
9	" G.S.	39 6/12ths
10	" B.D.	42 6/12ths
11	" E.J.	43 11/12ths
12	" S.M.	42 3/12ths
13	" K.R.	42 3/12ths
14	" C.L.	42 1/12th

Group Average Age = 42.61

## INTRODUCTION

It was decided that, before proceeding  
to more scientifically accurate methods of  
notation of various activities following systems  
involving, and even depending, on records  
of which from the subjective point of view.

It has long been known that the same  
frequency accompanied by a variety of other  
typical examples of the same nature with "hyper-  
and disease". THE FIRST INVESTIGATION

The investigation conducted by the group in  
collaboration with the author, and the results  
of several of the experiments conducted during  
the investigation in the course of the experiment  
conducted. (See also 1937, Bishop 1937)  
The results of the investigation have been observed with  
reference to the observed subject. It has been  
found that the results of the investigation  
of the investigation of the investigation of the investigation

I N T R O D U C T I O N

It was decided that, before proceeding to more objective and more scientifically accurate methods of investigating the problem of ovarian activity following hysterectomy, it might be interesting and even rewarding, to compare the three groups of women from the subjective aspect.

It has long been realised that the menopause is frequently accompanied by a variety of symptoms of which a typical example is the vasomotor disturbances known as "hot flushes". That these are very probably related to a fall in oestrogen production by the ovary is suggested by the well-known fact that replacement therapy in the form of natural or synthetic oestrogens very frequently effects a diminution in the severity of the symptom, if not its complete abolition. (Hawkinson 1938, Bishop 1947, Kupperman 1953). A similar response has been observed when oestrogens are given to the castrated subject. (Bishop 1938).

That relief from menopausal symptoms may, on the administration of oestrogens, proceed simultaneously with maturation of the cells of the postmenopausal vaginal epithelium/

epithelium has been reported by Greenblatt and his colleagues. (1950).

The vaginal epithelium, as will be shown later, may be regarded as a useful indicator of the blood's oestrogenic content. It is significant, therefore, that oestrogen replacement therapy which can convert an atrophic post-menopausal vaginal epithelium to a vaginal epithelium which closely resembles, if it is not actually indistinguishable from, that of a woman during reproductive life, very frequently produces simultaneous relief from those symptoms generally regarded as menopausal.

Although writers are not unanimous concerning which symptoms are to be regarded as distinctively menopausal, there is a considerable measure of agreement on the subject. An analysis of the menopause in one thousand healthy British women carried out by the Council of the Medical Women's Federation indicated that 62.3% of those interrogated experienced hot flushes, and that headaches, vertigo and nervous instability were the symptoms which occurred next in order of frequency. (Medical Women's Federation 1931).

These findings merely give statistical confirmation to the symptomatology of the menopause as it has long been described in many gynaecological textbooks.

PREVIOUS INVESTIGATIONS

Basing his conclusions on a survey of more than 300 women, all of whom were under the age of forty-five at the time when hysterectomy was performed, Richards (1951) found that, within two years of operation, 27% of those whose ovaries had been conserved had already developed hot flushes, and that 52% of those from whom one ovary had been removed showed this symptom. Of those subjects on whom bilateral oophorectomy had been performed however, no fewer than 98% had experienced hot flushes, the symptom usually showing itself within a few weeks of the removal of the ovaries.

If the hot flush is to be regarded as a symbol of the ovary's decline in activity, the above figures suggest that when the ovaries are preserved at operation they continue to function, in the majority of cases, for at least two years. That 27% of those whose ovaries were conserved experienced vasomotor symptoms within the next two years does not however, even in these cases, constitute indisputable evidence of a premature menopause, since the survey conducted by the Medical Women's Federation, of which mention has already been made, disclosed that 8% of the women who were the/



the subjects of this investigation reached a physiological menopause before the age of forty, and Kreiger (quoted by Kretschmar 1935) whose conclusions were based on the experience of 2,991 women, found that a spontaneous menopause occurred between the ages of thirty-six and forty in 12% of his subjects.

On this question it is appropriate to recall the work of Sessums and Murphy (1932) who found that 43.9% of 91 patients who had undergone the operation of hysterectomy with conservation of one or both ovaries, developed hot flushes before the age of forty. Although this series is small, one cannot ignore the fact that the apparent incidence of ovarian decline in function before the age of forty is several times as high as one would expect if no operation had been performed. It is necessary to note, however, that hot flushes may precede cessation of the menstrual flow, (Hendry 1936, Hawkinson 1938, Bishop 1947) so that the two phenomena here compared are not quite alternative manifestations of the same endocrinological change.

The influence of hysterectomy and oophorectomy on libido has been examined by a number of workers (Kretschmar and Gardiner/

Gardiner 1934, Richards 1951) but the results do not suggest an invariable or even a close relationship between oophorectomy and loss of libido.

#### M E T H O D

In order to compare the three groups of women who formed the subjects of this investigation, it was decided initially to make use of what the American endocrinologist Kupperman and his co-workers (1953) call the "menopausal index". This may be regarded as an expression in mathematical terms, obtained by means of a points system, of the severity of those symptoms which collectively comprise the menopausal syndrome.

The menopausal index of a woman is estimated in the following manner. The severity factor for each of a number of symptoms is individually determined. If the symptom is found to be present in a very pronounced form the maximum severity factor of three is recorded; if it occurs in moderate severity the factor will be two; and if it is present only in mild form the severity factor is represented by the figure one. If the symptom/

symptom is not present the severity factor is, of course, nil.

Since the vasomotor phenomenon known as the "hot flush" is considered to be a peculiarly significant symptom of the menopause the figure recorded for the severity factor is multiplied by a weighted factor of four, in order to give the conversion factor for that particular symptom.

Thus, if this symptom is thought to be manifest in moderate degree, a severity factor of two should be recorded, and this, when multiplied by the weighted factor of four, gives a conversion factor of eight.

Parasthesia, insomnia and "nervousness" are considered by Kupperman and his colleagues to rank next in significance in the list of menopausal symptoms, and each of these carries a weighted factor of two. Then come mental depression, vertigo, asthenia, arthralgia and myalgia (considered as one symptom), headaches, palpitation and formication, none of which is considered to be especially significant, so that the weighted factor for each of these is one. The sum of the conversion factors for each of her symptoms gives the menopausal index of a particular patient.

Now, it is very doubtful whether the above symptoms would find/

find general acceptance as those which are most typical of the menopause. One writer's list, it would appear, differs slightly from that of another.

It might also justifiably be objected that this method of estimating the severity of a menopause falls short of accuracy in that some of the listed symptoms, e.g. headache, arthralgia and myalgia could be produced by a great variety of conditions of which the menopause is only one, and not by any means the most probable one. Since, however, the main symptoms are "weighted" and the element of error is not likely to influence the results of one group more than another, it may be argued that even with its admitted inaccuracies, the menopausal index, when employed for comparative purposes, furnishes a useful, if somewhat crude, indication of the relative severity of the menopausal syndrome. It was decided, therefore, that the menopausal index of each woman in all three groups should be determined, and comparisons made thereafter between the average figures of each group.

Since the vasomotor phenomenon known as the "hot flush" is probably the most characteristic, as well as the most common/

common, menopausal symptom, the numerical values entered against it have been separately tabulated, so that comparisons of the relative severity of this symptom might be made between the three different groups of women who form the subjects of this investigation.

One word of explanation is now necessary. It may seem illogical to write about the menopausal index of women, who, like those of group (c) are known to be premenopausal, since this entire group, it will be remembered, consists of subjects who, by definition, still continued to menstruate.

The value of this investigation, however, consists in its demonstration of similarities or differences between the three groups of subject. It seemed desirable, therefore, that the women of group (c) should be asked questions akin to those which were put to the subjects of groups (a) and (b), so that, for purposes of comparison, one might obtain what, for want of a better term and somewhat in defiance of logic, one will designate as the menopausal index of this group.

As the subjects of group (c) had undergone no operation, it was necessary to modify the wording of the questions.

One/

One could not, for example, ask if a particular symptom had presented itself "since operation" so the phrase "within the past two years" was substituted.

1	1/1/1948	F	1	1/1/1948	1
2	1/1/1948	F	2	1/1/1948	2
3	1/1/1948	F	3	1/1/1948	3
4	1/1/1948	F	4	1/1/1948	4
5	1/1/1948	F	5	1/1/1948	5
6	1/1/1948	F	6	1/1/1948	6
7	1/1/1948	F	7	1/1/1948	7
8	1/1/1948	F	8	1/1/1948	8
9	1/1/1948	F	9	1/1/1948	9
10	1/1/1948	F	10	1/1/1948	10

Summary of symptoms since operation

1	1/1/1948	F	1	1/1/1948	1
2	1/1/1948	F	2	1/1/1948	2
3	1/1/1948	F	3	1/1/1948	3
4	1/1/1948	F	4	1/1/1948	4
5	1/1/1948	F	5	1/1/1948	5
6	1/1/1948	F	6	1/1/1948	6
7	1/1/1948	F	7	1/1/1948	7
8	1/1/1948	F	8	1/1/1948	8
9	1/1/1948	F	9	1/1/1948	9
10	1/1/1948	F	10	1/1/1948	10

Summary of symptoms since operation - 1/1/1948  
 Summary of symptoms since operation - 1/1/1948  
 Summary of symptoms since operation - 1/1/1948  
 Summary of symptoms since operation - 1/1/1948

RESULTSGroup (a) - Table IVCases of Hysterectomy with Conservation of Both Ovaries

<u>No.</u>	<u>Initials</u>	<u>Age</u> <u>Yrs.</u>	<u>Total or</u> <u>Subtotal</u> <u>Opera-</u> <u>tion</u>	<u>Interval</u> <u>since</u> <u>Opera-</u> <u>tion</u>	<u>Meno</u> <u>pausal</u> <u>Index</u>	<u>Index of</u> <u>Vasomotor</u> <u>Symptoms</u>	<u>Libido</u> <u>since</u> <u>Opera-</u> <u>tion</u>
1a	Mrs. B.D.	40 6/12ths	T	3 yrs.	0	0	No change
2a	" M.M.	42 4/12ths	T	1 1/12th	14	0	Decrease
3a	" J.F.(D)	48 6/12ths	T	1 7/12ths	2	0	No change
4a	" E.D.	46 3/12ths	T	1	0	0	Decrease
5a	" J.F.(B)	45 10/12ths	T	1 6/12ths	11	0	No change
6a	" J.C.(K)	42 9/12ths	T	2 10/12ths	1	0	Do.
7a	" M.McD	43 6/12ths	S	3	0	0	Do.
8a	" J.C.(E)	36 6/12ths	T	2 4/12ths	2	0	Do.
9a	" M.H.(C)	37 1/12th	T	2 2/12ths	0	0	Do.
10a	" H.T.	40 3/12ths	T	1 6/12ths	12	0	Do.
11a	" C.K.	38 5/12ths	S	1	3	0	Increase
12a	" A.R.	46 5/12ths	T	1 7/12ths	8	0	No change
13a	" M.H.(T)	41 1/12th		6 6/12ths	14	4	Decrease
14a	" J.S.	39 6/12ths	T	1 10/12ths	4	0	No change
15a	" I.R.	45 1/12th	S	1 6/12ths	6	0	Do.
16a	" J.McI	48	T	1	14	0	Decrease

Cases of Hysterectomy with Conservation of One Ovary

17a	" M.W.	33 2/12ths	S	1 1/12th	4	0	Increase
18a	" H.P.	45 8/12ths	T	1 3/12ths	2	0	Do.
19a	" E.C.	46	S	1 10/12ths	0	0	No change
20a	" J.O'N	40	S	1	0	0	Do.
21a	" M.D.	48 4/12ths	T	1 3/12ths	14	4	Decrease
22a	" J.B.	40 2/12ths	T	4 11/12ths	14	8	No change
23a	" M.S.	51	T	1 10/12ths	4	4	Do.
24a	" M.C.	41 3/12ths	S	6 6/12ths	13	8	Decrease

Group Average Age = 42.81

Group Average Menopausal Index = 5.92 (S.D. = 5.57)\*

Group Average Index for Vasomotor Symptoms = 1.17 (S.D. = 2.45)\*

Percentage showing Vasomotor Symptoms = 20.83%

Average Interval since Operation = 2.2 years.

\* S.D. = Standard Deviation

Group (b) - Table VCases of Hysterectomy with Removal of Both Ovaries

<u>No.</u>	<u>Initials</u>	<u>Age</u> <u>Yrs.</u>	<u>Total or</u> <u>Subtotal</u> <u>Opera-</u> <u>tion</u>	<u>Interval</u> <u>since</u> <u>Opera-</u> <u>tion</u>	<u>Meno</u> <u>pausal</u> <u>Index</u>	<u>Index of</u> <u>Vasomotor</u> <u>Symptoms</u>	<u>Libido</u> <u>since</u> <u>Opera-</u> <u>tion</u>
1b	Mrs. A.S.	42	T	1 7/12ths	26	12	Decrease
2b	" A.Y.	44	T	1 1/12th	22	12	No change
3b	" A.C.	45 5/12ths	T	1 11/12ths	17	12	Increase
4b	" M.W.(C)	39 5/12ths	S	9	12	4	Decrease
5b	" H.W.	47 6/12ths	T	2	36	12	Do.
6b	Miss B.F.	49	T	1 4/12ths	22	4	No change
7b	Mrs. M.B.	42	T	1 1/12th	26	12	Do.
8b	" J.S.	48 6/12ths	T	1 11/12ths	18	12	Decrease
9b	" M.S.(C)	50 6/12ths	T	1	2	0	Do.
10b	" A.H.	39 11/12ths	T	1	26	12	Do.
11b	" M.S.(K)	44 2/12ths	T	1	27	12	No change
12b	" C.O'G	41 5/12ths	T	2 7/12ths	24	12	Decrease
13b	" M.P.	41 4/12ths	S	2 2/12ths	27	12	Do.
14b	" P.F.	30	T	1	16	12	No change
15b	" A.T.	33 6/12ths	S	1 9/12ths	36	12	Decrease
16b	" M.D.	41 8/12ths	S	1 4/12ths	10	8	Do.
17b	" C.P.	48 1/12th	S	1 4/12ths	39	12	Do.
18b	" M.W.(D)	43	T	1 1/12th	16	8	Do.
19b	" E.M.	45 5/12ths	S	1 5/12ths	3	0	Do.
20b	" A.W.	46 7/12ths	T	1 7/12ths	4	0	No change
21b	Miss J.McG	43 2/12ths	T	1 7/12ths	4	0	Do.
22b	" A.E.	49 8/12ths	S	1 7/12ths	16	12	Decrease
23b	Mrs. E.Y.	51	T	1 11/12ths	29	12	Do.
24b	" E.H.	46 5/12ths	T	1 5/12ths	32	12	Do.

Group Average Age = 43.88 years

Group Average Menopausal Index = 20.42 (Standard Deviation = 10.41)

Group Average Index for Vasomotor Symptoms = 9 (Standard Deviation 4.65)

Percentage showing Vasomotor Symptoms = 79.58

Group Average Interval since Operation = 1.82 years



Group (c) - Table VIWomen who had undergone neither Hysterectomy nor Oophorectomy

<u>No.</u>	<u>Initials</u>	<u>Age</u> <u>Yrs.</u>	<u>Meno</u> <u>pausal</u> <u>Index</u>	<u>Index of</u> <u>Vasomotor</u> <u>Symptoms</u>	<u>Libido</u> <u>during</u> <u>last 2</u> <u>years</u>
1c	Mrs. W.D.	40 1/12th	0	0	No change
2c	" S.D.	43 4/12ths	0	0	Decrease
3c	" M.R.	43	3	0	No change
4c	" D.R.	38 6/12ths	0	0	Increase
5c	" J.K.	41, 5/12ths	4	0	No change
6c	" J.H.	40	0	0	Do.
7c	" C.W.	48 7/12ths	0	0	Do.
8c	" N.McG	49 2/12ths	16	0	Decrease
9c	" G.S.	39 6/12ths	0	0	No change
10c	" B.D.	42 6/12ths	5	4	Decrease
11c	" E.J.	43 11/12ths	0	0	No change
12c	" S.M.	42 8/12ths	1	0	Decrease
13c	" K.R.	42 3/12ths	4	0	No change
14c	" C.L.	42 1/12th	0	0	Do.

Group Average Age = 42.61

Group Average Menopausal Index = 2.36 (S.D. 4.18)\*

Group Average Index for Vasomotor Symptoms = .28. (S.D. .92)\*

\* S.D. = Standard Deviation.

Table No. VII

Comparing the Menopausal Indices of groups (a) and (b)  
and illustrating their statistical significance.

Menopausal Index of group (a)	Mean Value	5.92 (Standard Deviation
" " " (b)	" "	20.42 (Standard Deviation
		5.57)
		10.41)
Standard error of the means		2.704
Difference of the means		14.5
<u>Difference of the means</u>	=	5.4
<u>Standard error of the means</u>		

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Table No. VIII

Comparing the Menopausal Indices of groups (a) and (c)  
and illustrating their statistical significance.

Menopausal Index of group (a)	Mean value	5.92 (Standard Deviation
" " " (c)	" "	2.36 (Standard Deviation
		5.57)
		4.18)
Standard error of the means		1.71
Difference of the means		3.56
<u>Difference of the means</u>	=	2.08
<u>Standard error of the means</u>		

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Table No. IX

Comparing the Indices for Vasomotor Symptoms of groups (a) and (b) and illustrating their statistical significance.

Index for Vasomotor Symptoms	(Mean Value)	Group (a)	1.17
	(Standard Deviation)		2.45)
"	"	"	(Mean Value) Group (b) 9
			(Standard Deviation 4.65)
Standard error of the means	=		1.15
Difference of means	=		7.83
<u>Difference of means</u>			
Standard error of means	=		6.81

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Table No. X

Comparing indices of Vasomotor Symptoms of groups (a) and (c) and illustrating their statistical significance.

Index for Vasomotor Symptoms	(Mean Value)	Group (a)	1.17
	(Standard Deviation)		2.45)
"	"	"	(Meal Value ) Group (c) .28
			(Standard Deviation .92)
<u>Standard Error of the means</u>	=		.68
<u>Difference of means</u>	=		.89
<u>Difference of means</u>			
Standard error of means	=		1.31

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Table No. XI

Showing Average Menopausal Indices of the Three Groups of Subject in this Investigation.

Average Menopausal Index in group	(a)	5.92
"	(b)	20.42
"	(c)	2.36

Table No. XII

Showing Average Indices for Vasomotor Symptoms of the Three Groups of Subject in this Investigation.

Average Index for Vasomotor Symptoms of Group	(a)	1.17
"	(b)	9.00
"	(c)	.28

Table No. XIII

Showing Number and Percentage of Women of each group exhibiting Vasomotor Symptoms.

	Number in Group	No. showing Vasomotor Symptoms	% showing Vasomotor Symptoms
Group (a)	24	5	20.83%
" (b)	24	19	79.58%
" (c)	14	1	7.14%

Table No. XIVThe Libido in Relation to Hysterectomy

	No. of Women with increased Libido since Hysterectomy	No. of Women with decreased Libido since Hysterectomy	No. of Women with no change in Libido since Hysterectomy
Group (a)	3 (12.5%)	6 (25%)	15 (62.5%)
Group (b)	1 (4.17%)	16 (66.66%)	7 (29.17%)
	<u>Changes in Libido during previous 2 years</u>		
	<u>Increased</u>	<u>Decreased</u>	<u>No change</u>
Group (c)	1 (7.14%)	4 (28.57%)	9 (64.28%)

DISCUSSION

The results of this investigation would appear to indicate that, when hysterectomy is performed on premenopausal women, the symptomatic manifestations of the menopause are, on the whole, much less pronounced in those patients whose ovaries have been conserved than in those whose ovaries have been removed.

Not only is the average menopausal index of the oophorectomised group (20.42) much higher than that of the group whose ovaries were not removed at operation (5.92), but it is to be noted that, whereas almost four women out of every five (79.58%) of the former group complained of vasomotor symptoms, only about one in five (20.83%) of the latter group had suffered from this complaint.

Although the group average menopausal index of those women who had undergone the operation of hysterectomy with conservation of one or both ovaries (5.92) is higher than the average menopausal index of that group who had undergone no operation (2.32), it is noteworthy that even this small group of fourteen women contained one subject (No. 10c Mrs. B.D.) who, though continuing to menstruate, complained of "hot flushes".

It must also be remembered that every woman of the latter group had, within the two years immediately preceding this investigation, given birth to a child. Group (c), though it may be said to represent the normal woman of that particular age distribution, also represents what, regarded from the aspect of ovarian activity, must be considered a better-than-average sample of the female population, since severe ovarian dysfunction would be much less likely to be encountered in this group than in a cross-section of the female population of corresponding age, or even in those women who are the subjects of the other groups in this investigation.

That one subject (Miss M.S. No. 23a), of group (a) who had, at the time of this investigation, reached the age of 51, should have begun to complain of "hot flushes" is not surprising, and need not necessarily be linked in any cause and effect relationship with the operation of hysterectomy which she had undergone two years earlier.

More interesting is the fact that two women of group (a) Mrs. J.B. (No. 22a) and Mrs. M.C. (No. 24a) who were only aged 40 and 41 respectively when the investigation was conducted/

conducted, complained of "hot flushes". It should be noted, however, that in both cases the symptom was of recent onset, whereas the interval of time that had elapsed since operation was 4 years, 11 months in one instance and six and a half years in the other.

Even the most redoubtable protagonist of oophorectomy would therefore find it difficult to prove that these womens' ovaries had become prematurely inactive as a result of hysterectomy.

It is appropriate to recall that the survey conducted by the Council of the Medical Women's Federation (1933) indicated that about 8% of women reach a physiological menopause before the age of forty. Against the background of this piece of information the fact that 2 women out of a group of 24 (i.e. 8.3%) should begin to complain of "hot flushes" soon after attaining the age of 40, suggests that the operation of hysterectomy need not have been a causative factor in the development of their vasomotor symptoms.

Then, it has been suggested that where ovarian loss of function follows the operation of hysterectomy, the explanation may be that the blood supply to the ovary has been/



been unwittingly damaged by the surgeon (Aldridge 1950, Richards 1951). Were this so, however, in the cases just mentioned, one would have expected the symptoms indicative of ovarian failure to have become apparent much earlier than four to six years after the operation.

Another subject of group (a) who complained of having experienced "hot flushes" was Mrs. M.H. (No. 13a) who, six and a half years earlier, had undergone the operation of subtotal hysterectomy. This woman also reported that she "menstruated regularly for one or two days every four weeks". The accuracy of this statement was subsequently verified over a period of six months. As will be shown later, additional (and more objective) evidence strongly suggested that, for a period of two years after the time of this investigation, this woman's ovaries were still functioning. It would seem either that this patient mistook the nature of her symptoms or that the "hot flush" is a somewhat unreliable indicator of declining ovarian activity.

The fifth member of group (a) to complain of "hot flushes" also merits individual comment. This patient, Mrs. M.D. (No. 21a) who had undergone the operation of total hysterectomy/

hysterectomy with unilateral oophorectomy some fifteen months prior to the date of this investigation, stated, like the previous patient, that the symptom had its onset shortly after operation and had lasted for a few weeks only.

As will be shown later, the evidence of more objective tests suggests that it is at least feasible that, while in a state of postoperative debility, this woman had experienced some vasomotor disturbances which she has been unable to distinguish from the "hot flushes" of the menopausal subject.

It is not possible, therefore, to select from the 24 subjects of group (a), one convincing example of a woman who has suffered vasomotor disturbances which may, with a fair degree of probability, be logically linked with her hysterectomy.

While reiterating the fallibility of this investigation as a scientific test of continuing ovarian function, one must conclude that the general trend of its results has been to indicate that when hysterectomy with ovarian conservation is performed on a premenopausal woman, there is no convincing evidence that the operation will result in a premature cessation of ovarian activity.

SUMMARY and CONCLUSIONS

In this investigation of the problem of ovarian activity following hysterectomy, an attempt has been made to ascertain to what degree each subject of the main investigation group (a) and the two control groups (b) and (c) experienced those symptoms which collectively comprise the menopausal syndrome.

By this means it was hoped that some indication might be given of the state of ovarian activity (or its absence) in each individual of each group. Reference is made to similar investigations by Richards (1951) and Sessums and Murphy (1934).

For this purpose use was made of the "menopausal index". This may be regarded as an expression in mathematical terms of the relative severity of those symptoms usually associated with the menopause, as experienced by the individual. The index is calculated on a points system. The method of its use has been described and its limitations as an indicator of ovarian function emphasised. The menopausal index of each woman taking part in this investigation was determined, and also the average menopausal index of each group.

It is realised that if the results are to be considered as having any significance they must exhibit marked differences and indicate very definite trends. It is believed that these differences and trends may be observed in the various tables in which the results are shown (see Tables IV to XIII).

It was found that those women who had undergone the operation of hysterectomy with bilateral oophorectomy had a group average menopausal index of 20.42, whereas the average menopausal index of that group of women who had undergone the operation of hysterectomy with conservation of one or both ovaries was 5.92. The fact that the average menopausal index of the normal subjects of approximately comparable age was found to be 2.36 suggests a closer affinity, in terms of ovarian function, between the normal subjects and those who had undergone hysterectomy with some degree of ovarian conservation, than between this latter group and those who had undergone hysterectomy with bilateral oophorectomy. It has been emphasised that the normal group represents a section of the female population whose ovarian function must be regarded/

regarded as better than that of a mere cross-section of women of comparable age, since each of these women had given birth to a child within the past two years. The fact that only two subjects out of fourteen were over 44 years of age probably also helps to flatter the low average value of the menopausal index of this group as compared with that of those women who had undergone hysterectomy but still retained one or both ovaries. The indices for vasomotor symptoms of each woman and each group of women were also determined, and the results classified under separate headings (see Tables IV, V and VI).

Those women who had undergone hysterectomy with total or partial ovarian conservation were found to have an average index for vasomotor symptoms of 1.17 as compared with the normal group's average figure of .28. Those women from whom both uterus and ovaries had been removed were found to have a group average index for this symptom of 9.

These results emphasise the wide disparity in terms of symptomatology between those women who had been castrated and the two remaining groups.

Tables/

Tables IV and V also show that 79.85% of those women who had undergone hysterectomy with bilateral oophorectomy exhibited evidence of vasomotor symptoms, as compared with 20.83% of the group who had undergone hysterectomy with conservation of one or both ovaries. Among the normal group of women, 7.14% complained of hot flushes (see Table VI).

Reasons are given for believing that "hot flushes" cannot always be accepted as a reliable indicator of declining ovarian function.

The influence of hysterectomy and oophorectomy on libido was less clearly indicated in this investigation, though it is to be noted that whereas two thirds of those subjects who had lost both ovaries and uterus, stated that there had been an appreciable decline in libido following operation, only 25% of those who had undergone hysterectomy but still retained some ovarian tissue, made the same observation. Among the normal women of group (c), 28.57% had observed a considerable decline in libido during the two years immediately prior to this investigation.

The fact that in all three groups a certain percentage of women reported an increase in libido suggests, as has often been stated, that retention of sex feeling is not solely/

solely dependent on a functioning ovary.

The general trend of the results of this investigation is to indicate that there is no clear evidence that the operation of hysterectomy with ovarian conservation produces a premature cessation of ovarian activity.

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**SECOND INVESTIGATION**

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I N T R O D U C T I O N

The purpose of this investigation was to attempt, by means of daily temperature records, to deduce whether or not there was evidence of corpus luteum formation on the part of those ovaries which had been conserved at hysterectomy.

The test makes use of the fact that during the reproductive period of a woman's life the morning temperature, if taken with care while the subject is at rest, should, when charted daily, exhibit a significant rise, known as the "thermal shift" if, and about the time when, ovulation occurs.

The temperature of a regularly menstruating and ovulating woman may be said to follow a predictable pattern. From the onset of menstruation and during the oestrogenic phase of the cycle it remains at a relatively low level. Then, about the time of ovulation, it rises suddenly to a higher level at which it continues until just before menstruation, when it falls once more, and something like pre-ovulation figures are again recorded. The record may therefore be said to be diphasic in character.

### History of the "Thermal Shift"

A correlation between the cyclical variations in the basal body temperature and the events of the menstrual cycle was first made by Squire in 1868. His observation received further elaboration at the hands of Giles, who is recorded in the Transactions of the Obstetrical Society of London for 1897 as having stated that the temperature reached its lowest ebb in the middle of the intermenstrual period, gradually rising to its maximum two days before menstruation. A sudden drop, he claimed, was to be noted on the day preceding menstruation and a second slight drop at the end of the period. He added that a slow rise could be observed to occur during the first week following menstruation and a third fall at the beginning of the intermenstrual period.

Giles was unable to explain correctly the phenomenon which he had described with more than a fair degree of accuracy, and it was left to Fraenkel (1903) to relate the rise and subsequent fall in temperature to the life span of the corpus luteum, implying that this was the responsible agent, and offering as additional evidence the continued elevation that results when pregnancy occurs.

It/

It was subsequently demonstrated by various investigators, e.g. Kleitman (1933 (a) and (b)) and Vollman (1940), that men, postmenopausal women and girls who have not yet reached puberty, fail to exhibit this diphasic pattern in their daily temperature records.

Such observations, strengthened the theory that the rhythmic fluctuations which distinguish the temperature records of the ovulating subject are the manifestations of ovarian hormonal activity during the reproductive phase of a woman's life, and further support was to come from the work of Palmer and Devillers (1939) who showed that the injection of five milligrams of oestradiol benzoate into an oophorectomised woman was followed by a fall in temperature which lasted several days, and that the subsequent injection of ten milligrams of progesterone produced a rise in temperature.

That oestrogens exercise a depressing, and progesterone an elevating, effect on the basal body temperature is now generally accepted, the principle having been repeatedly confirmed by the experimental evidence of many observers (Buxton 1948, Davis 1948, Israel 1948, Nieburghs 1948, Perlman 1948).

These hormones, when administered to amenorrhoeic subjects in the reproductive period of life or to surgical

surgical castrates, act in accordance with the generalisation which has just been made, but it should also be mentioned that Magallon and Masters (1950) when studying a group of elderly postmenopausal women, made certain observations which suggest that the rule just enunciated may admit of modification when further research enlarges our knowledge of the hormonal processes involved. They discovered that when their subjects had been "primed" with oestradiol benzoate and kept on a maintenance dose of one milligram twice weekly, their temperature records exhibited no "thermal shift" after the administration of progesterone, but that progesterone, when given with no immediately antecedent administration of oestradiol, was capable of producing an elevation of temperature.

In explanation of these findings, Magallon and Masters postulate a certain relationship between the relative blood levels of progesterone and oestrogen as a necessary prerequisite of the sudden rise in the basal body temperature.

Such a theory might indeed account for the apparent discrepancy revealed by their investigations without in any way destroying the validity of the initial concept concerning the hyperthermic properties of progesterone.

Barton/

Barton and Wiesner (1945B) conducted the interesting experiment of injecting progesterone into women during the postovulatory phase of the cycle in order to discover what the effect would be on the already elevated temperatures of their subjects. They observed that while some responded with a further rise in temperature above 100° F., the remainder did not. Other workers, however, claimed to demonstrate a quantitative relationship between the degree by which the temperature rises and the dosage of progesterone given to the subject (Perlman 1948, Rothschild 1952).

The relationship between endogenous progesterone and the body's rise in temperature has been studied by several workers. Buxton and Engle (1950), who planned a number of laparotomies to be performed immediately the basal body temperature of the patient had exhibited what was assumed to be the "thermal shift", were not always able to report the finding of corpora lutea in the ovaries of their subjects. Greulich and his co-workers (1943), however, observed that when laparotomy was performed during the low temperature phase of the cycle no corpora lutea were revealed, whereas in each case in which the ovaries were removed at peak temperature level a corpus luteum was discovered.

The/

The evidence obtained by histological examination of endometrial biopsy has often been compared with that derived from the patient's temperature chart, and, in the overwhelming majority of cases, it has been found that the information supplied by either source concerning the ovarian cycle has been confirmed by the other (Martin 1943, Halbrecht 1945, Halbrecht 1947, Noyes 1950).

Rubenstein (1937) has reported complete agreement between the cyclical changes of the vaginal epithelium as revealed by vaginal smears and the pattern of the patient's temperature record.

#### The Mechanism of the "Thermal" Shift

In 1952 Rothchild and Allan published an article which demonstrated that, although the elevation of temperature produced by progesterone could not be influenced by the giving of oestrogens and androgens, yet, in some individuals, the administration of salicylates had an inhibitory effect upon it. Such a finding posed the possibility that the thermogenic activity of progesterone might/

might be mediated through a centre in the hypothalamus.

Then Elert (1951), observing that the elevation of temperature which he could produce in an amenorrhoeic woman by the administration of progesterone, was completely suppressed by the simultaneous administration of a central depressant in the form of .018 gram of Luminal, advanced the theory that progesterone exercises its influence on body temperature centrally through the medium of the diencephalon.

The question is still debated, but it is probably true to say that the consensus of informed opinion inclines towards the view that progesterone produces its effect on the basal body temperature through some mechanism involving the central nervous system.

#### The "Thermal Shift" as Evidence of Luteinisation

A multiplicity of evidence of which the preceding pages may be regarded as a brief synopsis, justifies the assumption that the elevation of temperature which has repeatedly been observed to occur in women of childbearing age/

age, about the time of ovulation, is produced by the action of endogenous progesterone.

The fact that the "thermal shift" is known to occur about the time of ovulation has been successfully used in the treatment of infertility (Davis 1948), and has been made the basis of an efficient form of contraception by Barton and Wiesner (1945A), who report that the period from the third day of the high-temperature phase of the cycle until menstruation may be regarded as "safe", in that they had instructed patients who did not desire to become pregnant to limit coitus to this part of the cycle, and had, up to the time of writing, encountered no failures. While the precise date of ovulation cannot be determined with complete finality on the basis of a woman's temperature chart (Siegler 1951), a diphasic temperature record may be said to constitute evidence of luteinisation, which in turn may be accepted as more than a fairly reliable indication that ovulation has occurred.

#### PREVIOUS INVESTIGATION

In a long and careful perusal of the literature there was/



was found only one attempt to ascertain, by the use of basal temperature records, whether or not the ovaries continue to show evidence of luteinisation following hysterectomy.

The sole published record of such an investigation would appear to be that of Davis and Fugo (1948) who found that, of twelve women whose ovaries had been conserved at hysterectomy and whose basal temperatures were recorded for four months following operation, four exhibited the diphasic curve which is presumed to denote luteinisation.

Such a report can hardly be considered an important contribution to the subject, since no information is given concerning the ages of the various patients, and no allowance is made for the possibility of a temporary disturbance of the body's hormonal relationships during the period immediately following operation.

M E T H O D

For this investigation each of the subjects in groups (a), (b) and (c) was instructed in the use of the clinical thermometer and asked to keep a record of her temperature for several months.

Many of these women had not previously used a thermometer, and although great care was taken to explain to them how it is read and how the results should be recorded, a number of the subjects in each group proved incapable of reading the instrument or of keeping a regular and reliable record. After a number of fruitless attempts to instruct them, it was therefore decided to dispense with the assistance of these subjects who took no further part in the investigation. In some cases the temperatures were taken and recorded by the subject's husband, or by one of her children who had learned to use a thermometer at school.

It was made clear to the subject by means of a typed sheet of instructions that the temperature ought preferably to be taken before she rose from bed in the morning, and that the thermometer should be retained in the mouth for at least/

least three minutes (measured by clock or watch).

As results obtained by the oral method are said to be as reliable as those obtained rectally or vaginally (Nieburgs 1946, Davis 1948), and as the method causes less difficulty and embarrassment to the subjects, no attempt was made to induce any of them to adopt either of these methods.

The necessity of taking the temperature at the same time each day was emphasised, as the basal body temperature of a woman has been shown to have a diurnal variation which is sufficiently great to obscure the diphasic pattern of the ovarian cycle if the time factor is ignored (Palmer 1950).

Each subject was also informed that no warm food or drink should be consumed just prior to the taking of the temperature, and that while it was being taken and for some time previously, she should be relaxed and at rest.

The subject was instructed, on removing the thermometer from her mouth, to read it and to record the reading at once, both on a chart which was provided, and by entering the date and the figure on a sheet of paper.

If the thermometer was lost or broken, the subject was to report the fact immediately by telephone, so that another might be provided. This occurred on dozens of occasions.

The records vary in duration from one month in the case of some of the subjects of group (c), to five months, and in several instances, the keeping of the record has been resumed after an interval of several months. The reason for this will be explained in the introduction to the next investigation.

RESULTS

Illustrative and significant portions of the temperature records of each subject of group (a) are here reproduced, showing the times at which 48-hour samples of urine were taken for pregnanediol estimations and the values obtained.

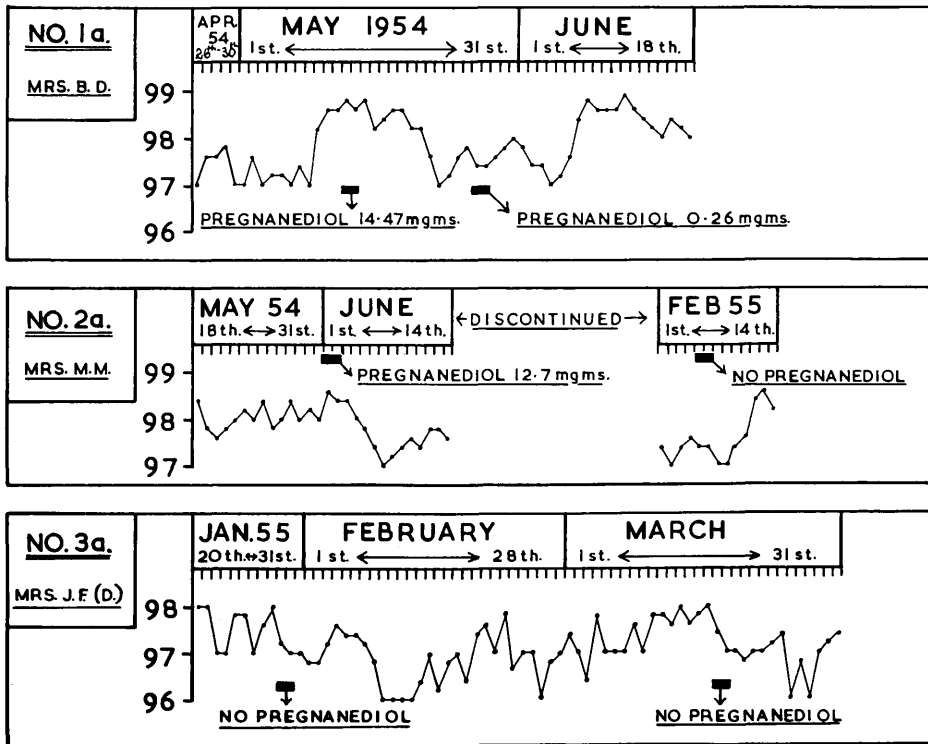


Fig. I showing significant portions of the temperature records of subjects 1a, 2a and 3a, and the results of urinary pregnanediol estimations in relation to temperature record

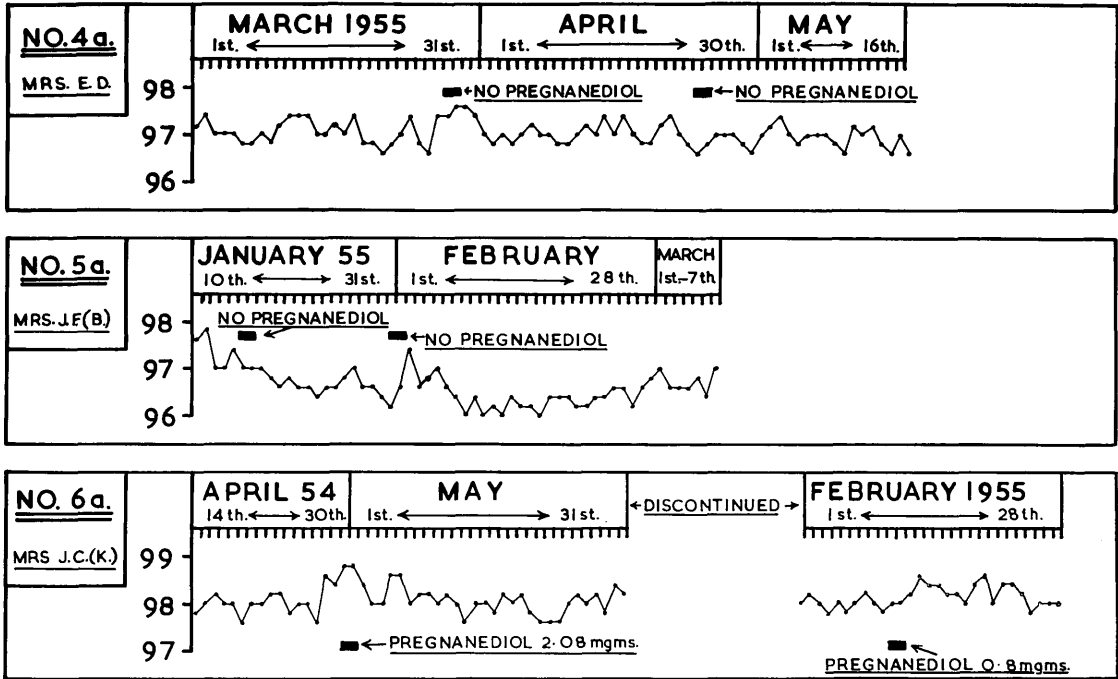


Fig. II showing illustrative portions of the temperature records of subjects 4a, 5a and 6a and the results of urinary pregnanediol estimations.

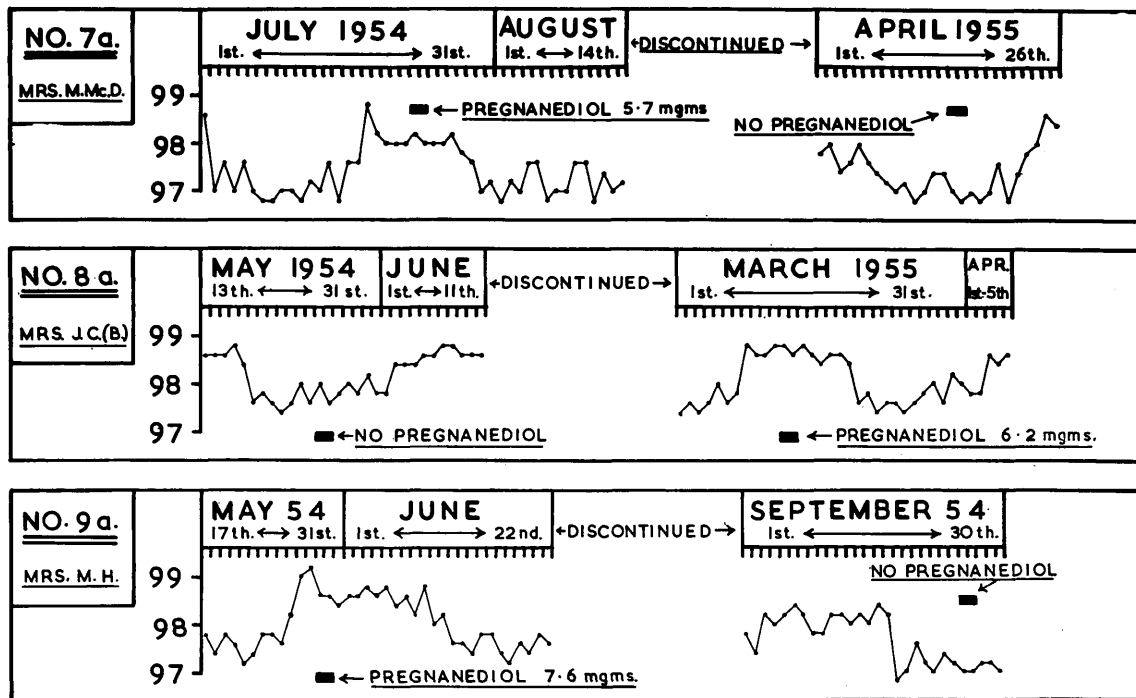


Fig. III showing illustrative portions of the temperature records of subjects 7a, 8a and 9a and the results of urinary pregnanediol estimations.

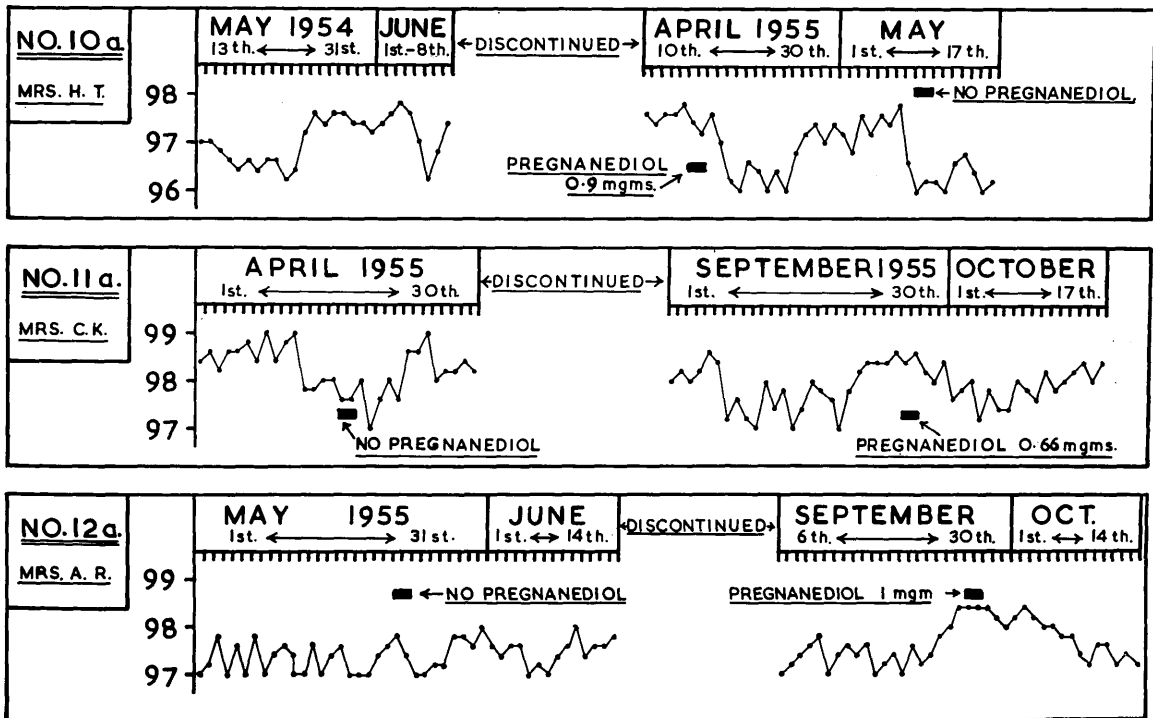


Fig. IV showing portions of the temperature records of subjects 10a, 11a and 12a and the results of urinary pregnanediol estimations.



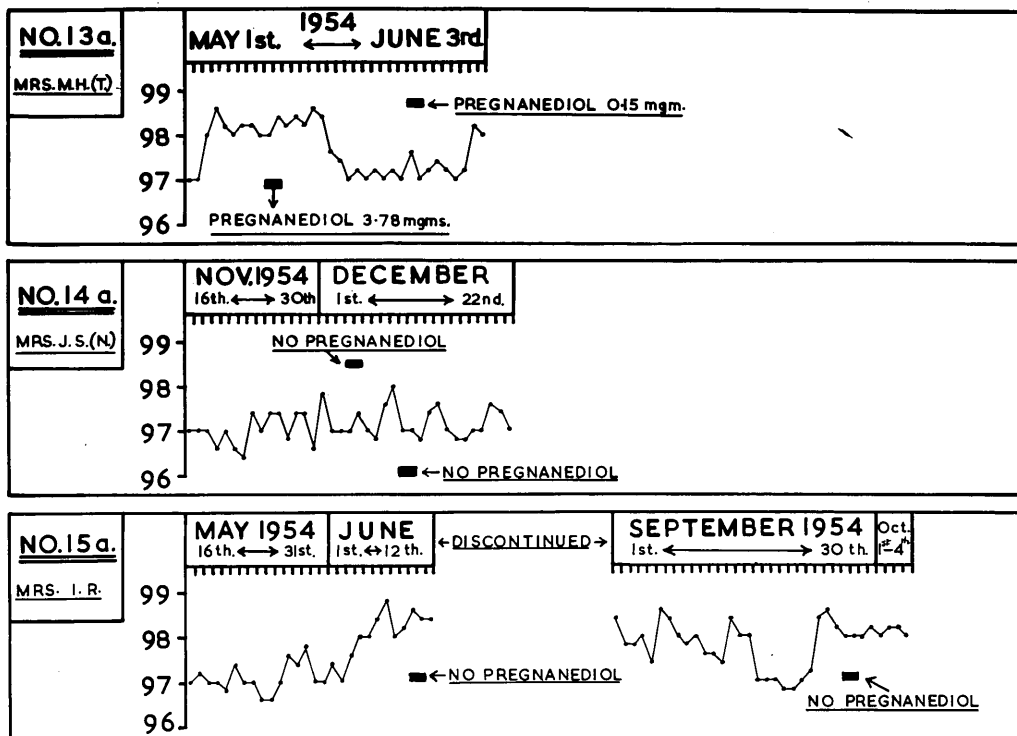


Fig. V showing portions of the temperature records of subjects 13a, 14a and 15a and the results of urinary pregnanediol estimations.

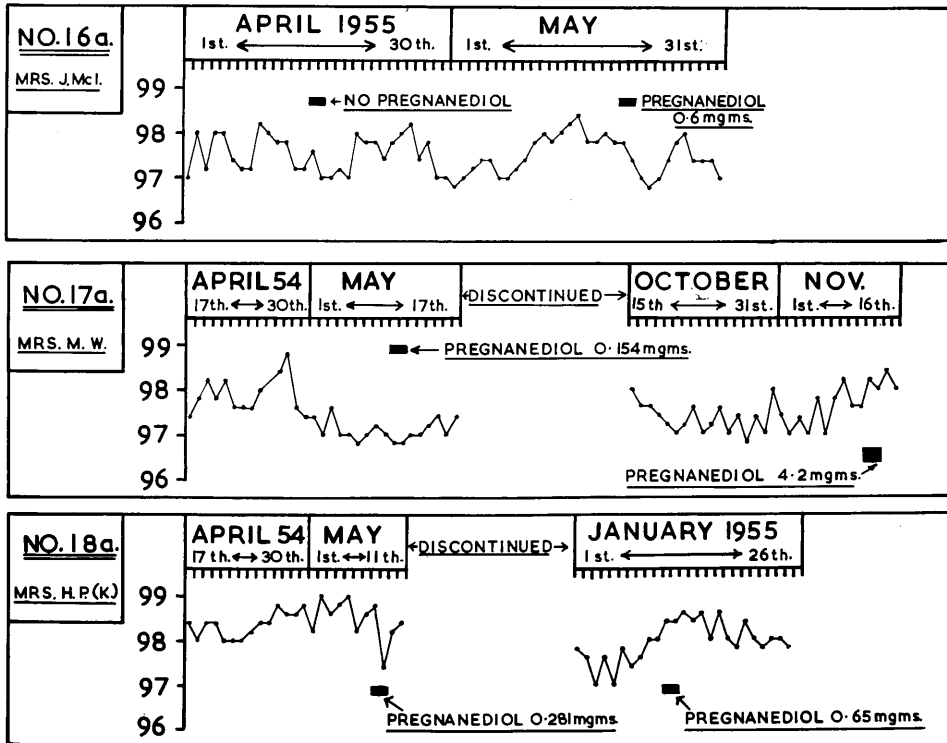


Fig. VI showing portions of the temperature records of subjects 16a, 17a and 18a and the results of urinary pregnanediol estimations.

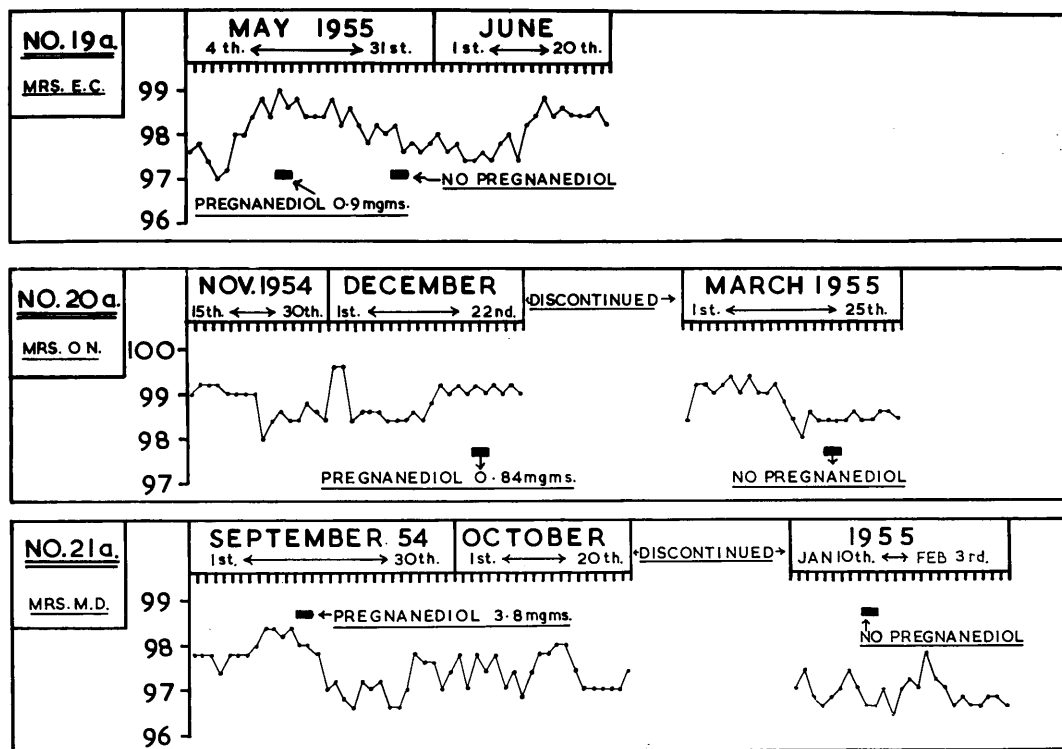


Fig. VII showing portions of the temperature records of subjects 19a, 20a and 21a and the results of urinary pregnanediol estimations.

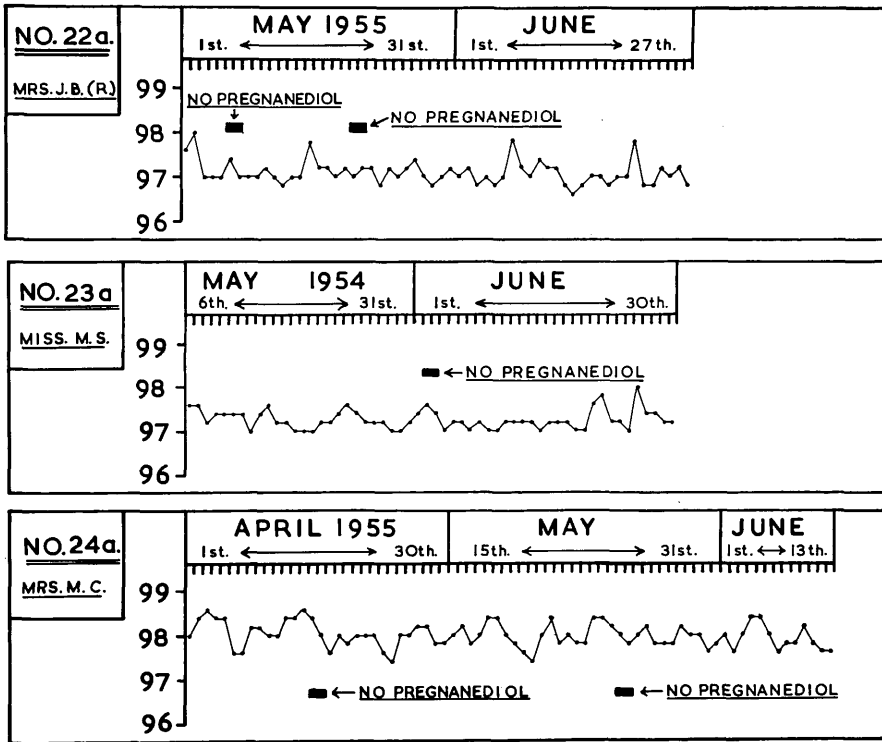


Fig. VIII showing portions of the temperature records of subjects 22a, 23a and 24a and the results of urinary pregnanediol estimations.

Characteristic portions of the temperature records of each subject of group (b) are here reproduced, showing also the dates on which 48-hour samples of urine were taken for pregnanediol estimation and the results obtained.

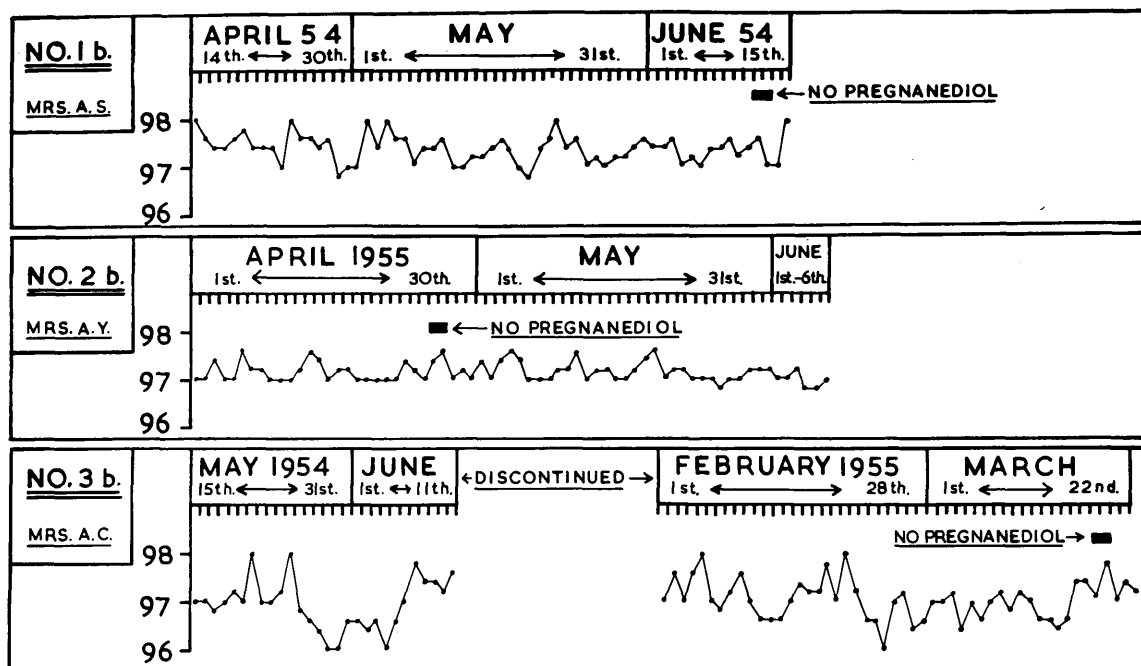


Fig. IX showing typical portions of the temperature records of subjects 1b, 2b and 3b and the results of urinary pregnanediol estimations.

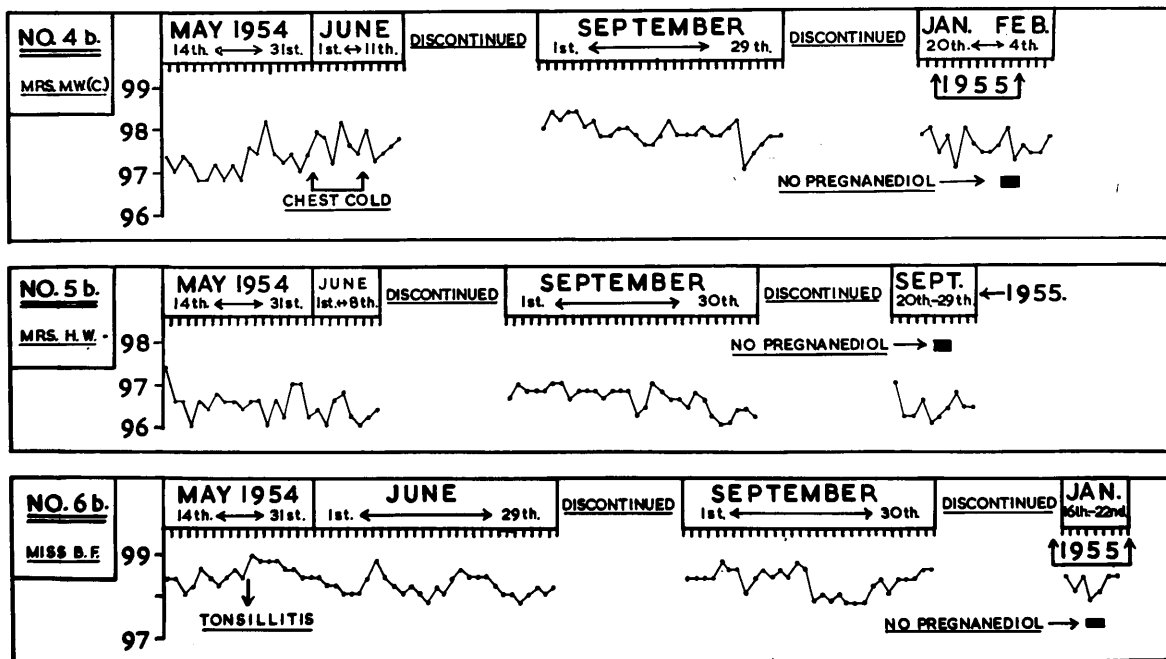


Fig. X showing typical portions of the temperature records of subjects 4b, 5b and 6b, and the results of urinary pregnanediol estimations

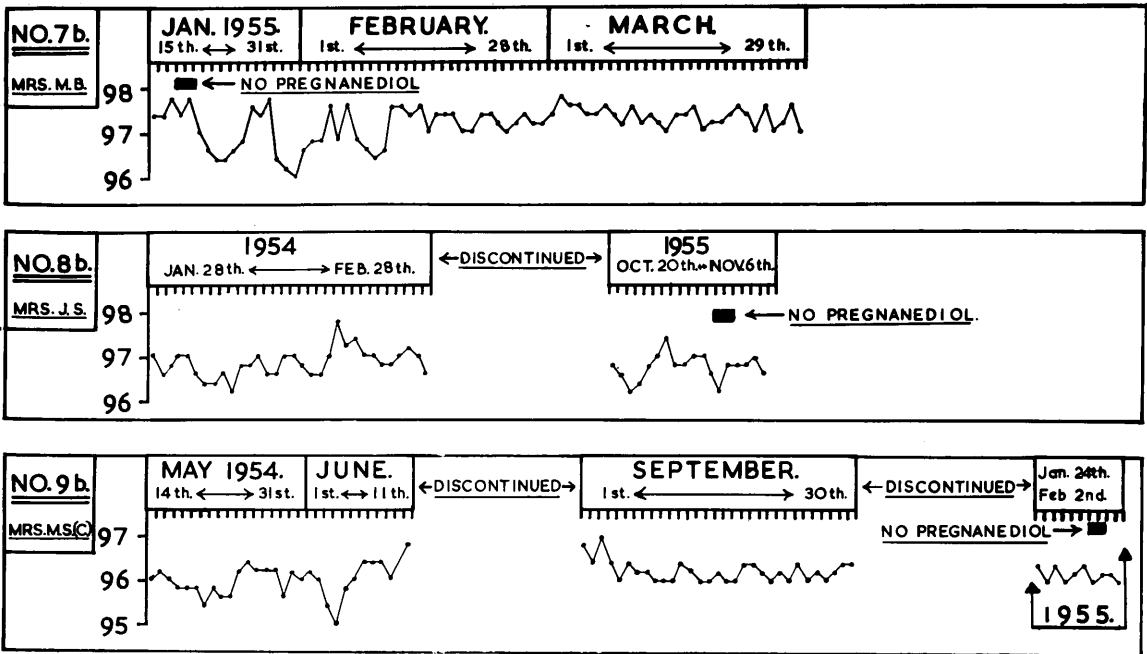


Fig. XI showing typical portions of the temperature records of subjects 7b, 8b and 9b and the results of urinary pregnanediol estimations.

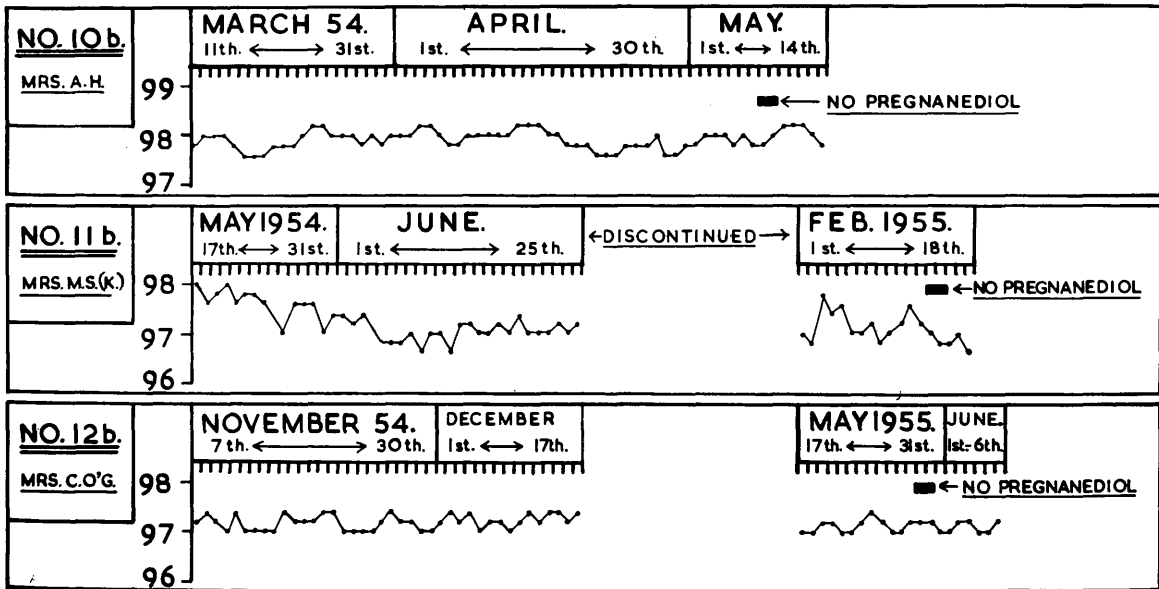


Fig. XII showing typical portions of the temperature records of subjects 10b, 11b and 12b and the results of urinary pregnanediol estimations.



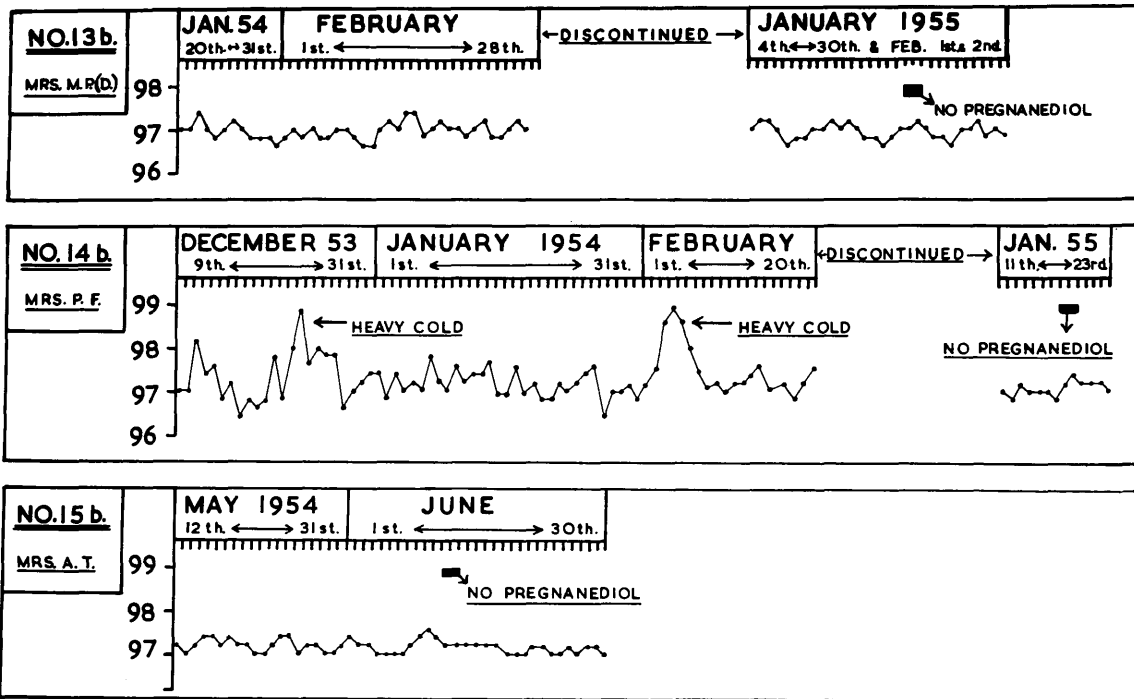


Fig. XIII showing typical portions of the temperature records of subjects 13b, 14b and 15b and the results of urinary pregnanediol estimations.

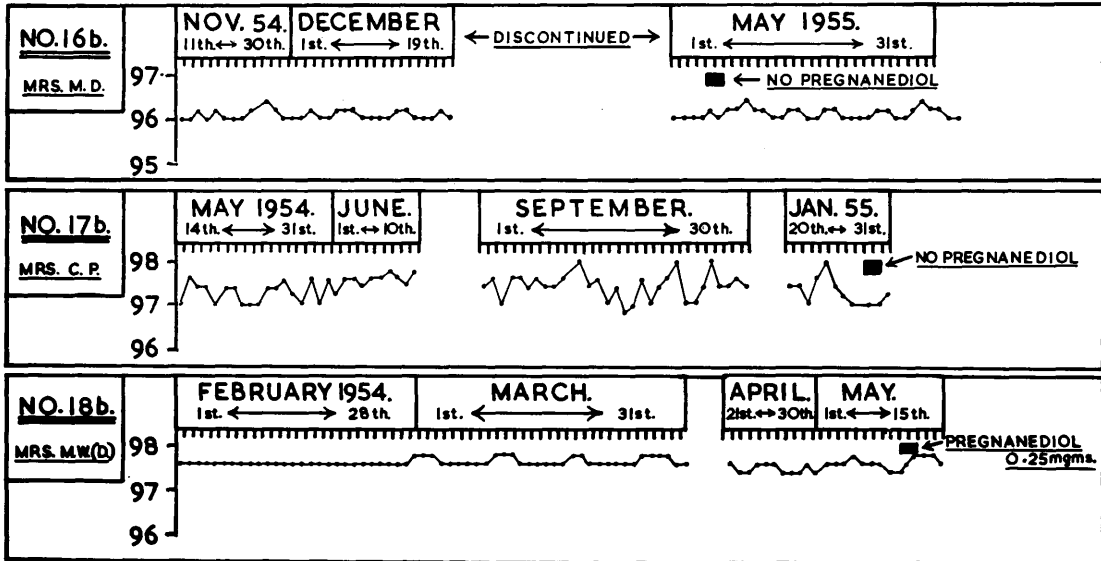


Fig. XIV showing typical portions of the temperature records of subjects 16b, 17b and 18b and the results of urinary pregnanediol estimations.

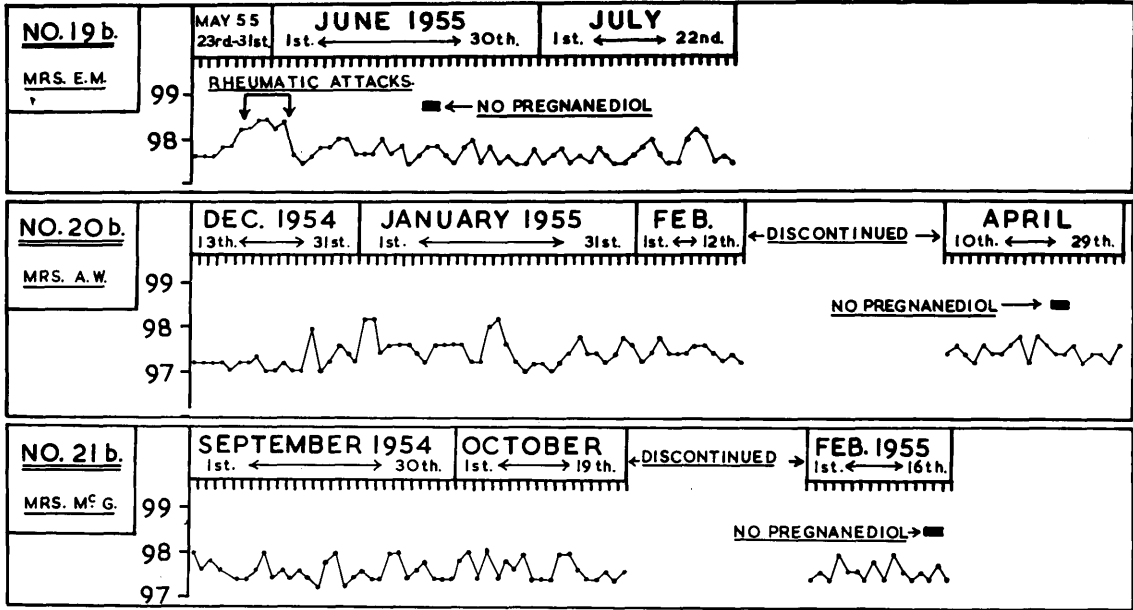


Fig. XV showing typical portions of the temperature records of subjects 19b, 20b and 21b and the results of urinary pregnanediol estimations.

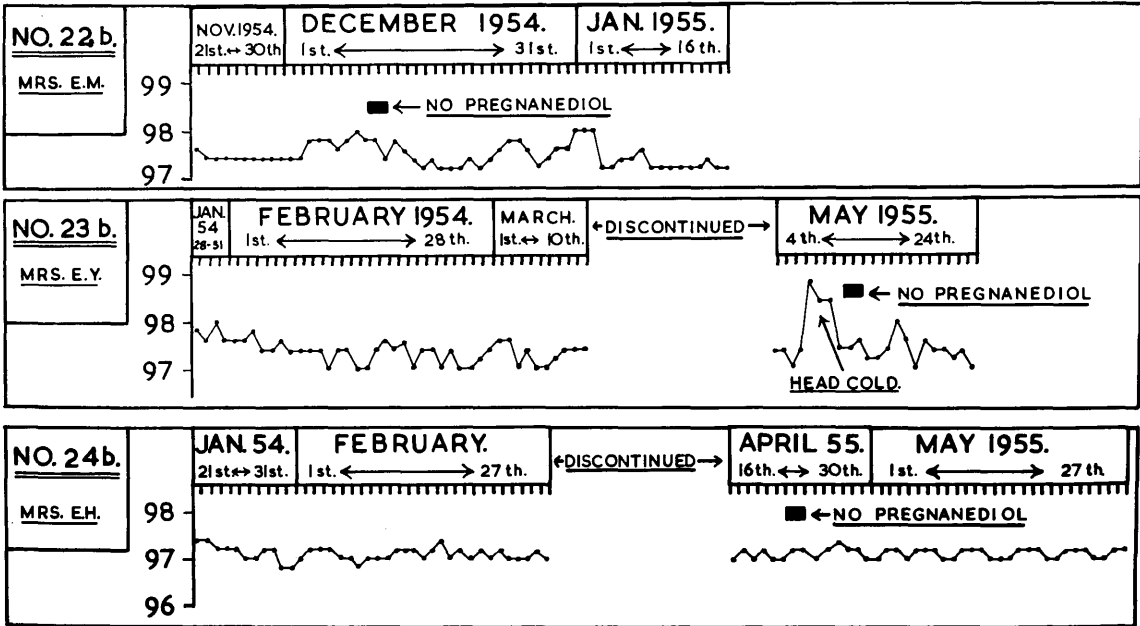


Fig. XVI showing typical portions of the temperature records of subjects 22b, 23b and 24b and the results of urinary pregnanediol estimations.

A temperature record of each woman of group (c) is now reproduced. It extends from one menstrual period to the next. The record also indicates the dates on which 48-hour samples of urine were taken for pregnanediol estimation and shows the results of these estimations. An endometrial biopsy was also obtained from the majority of the subjects of this group. The reproduced temperature record shows the date on which the biopsy was taken and includes a summary of its histological appearance.

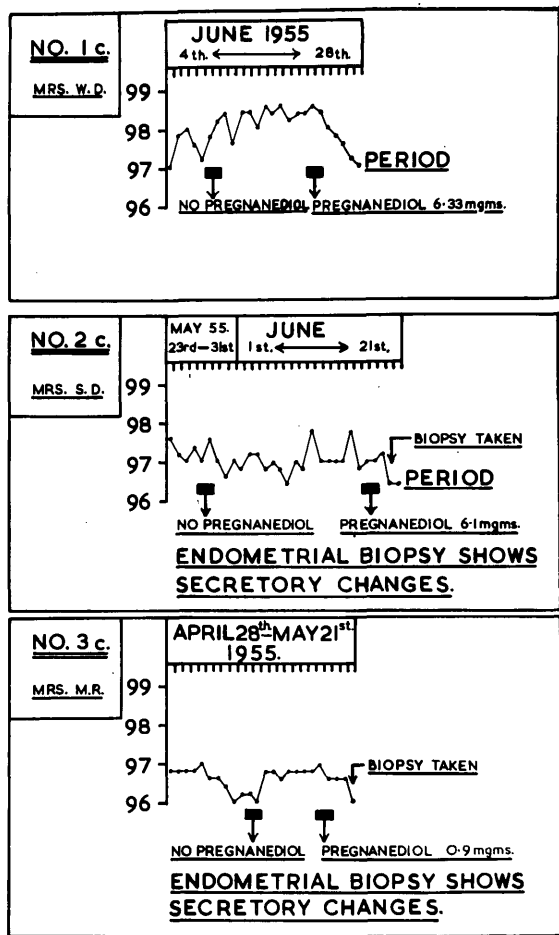


Fig. XVII showing the temperature records of subjects 1c, 2c and 3c with the results of pregnanediol estimations and endometrial biopsies.

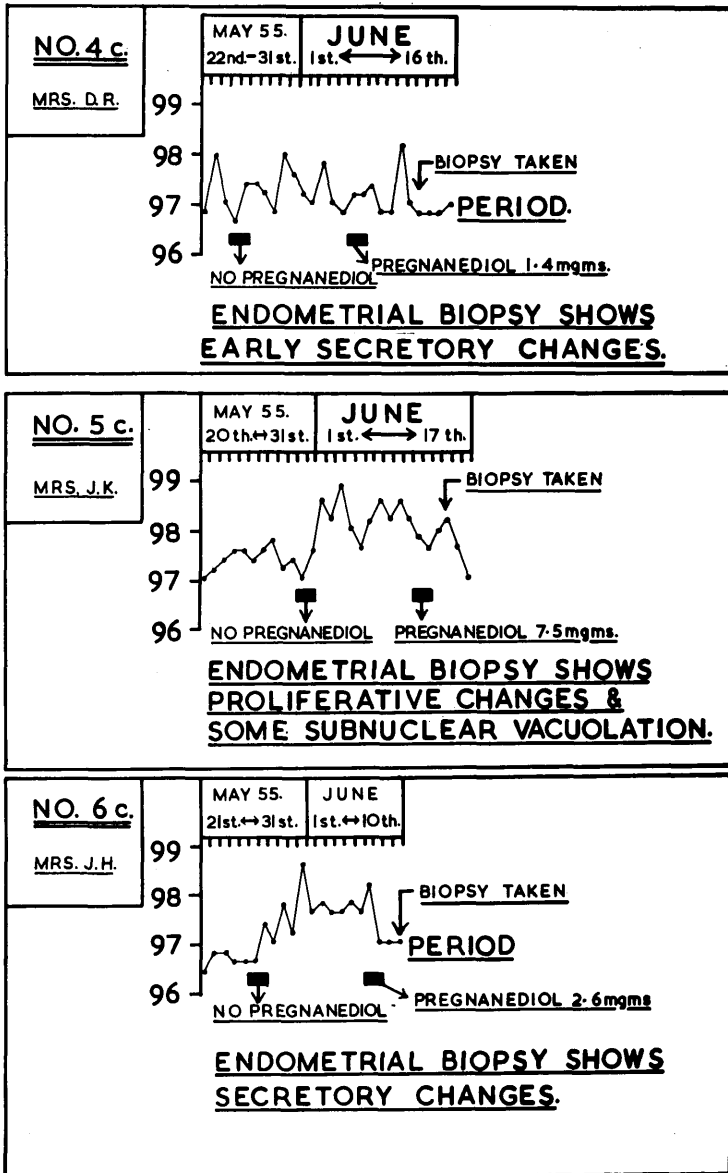


Fig. XVIII showing the temperature records of subjects 4c, 5c and 6c with the results of pregnanediol estimations and endometrial biopsies.

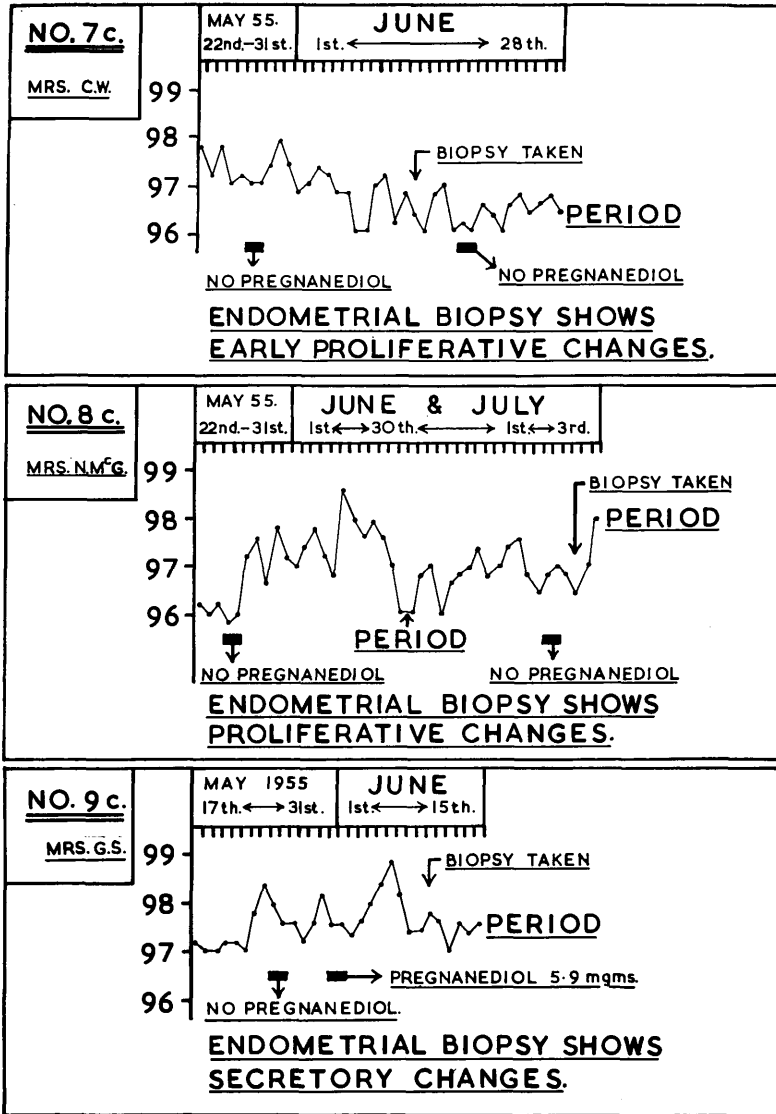


Fig. XIX showing the temperature records of subjects 7c, 8c and 9c with the results of pregnanediol estimations and endometrial biopsies.

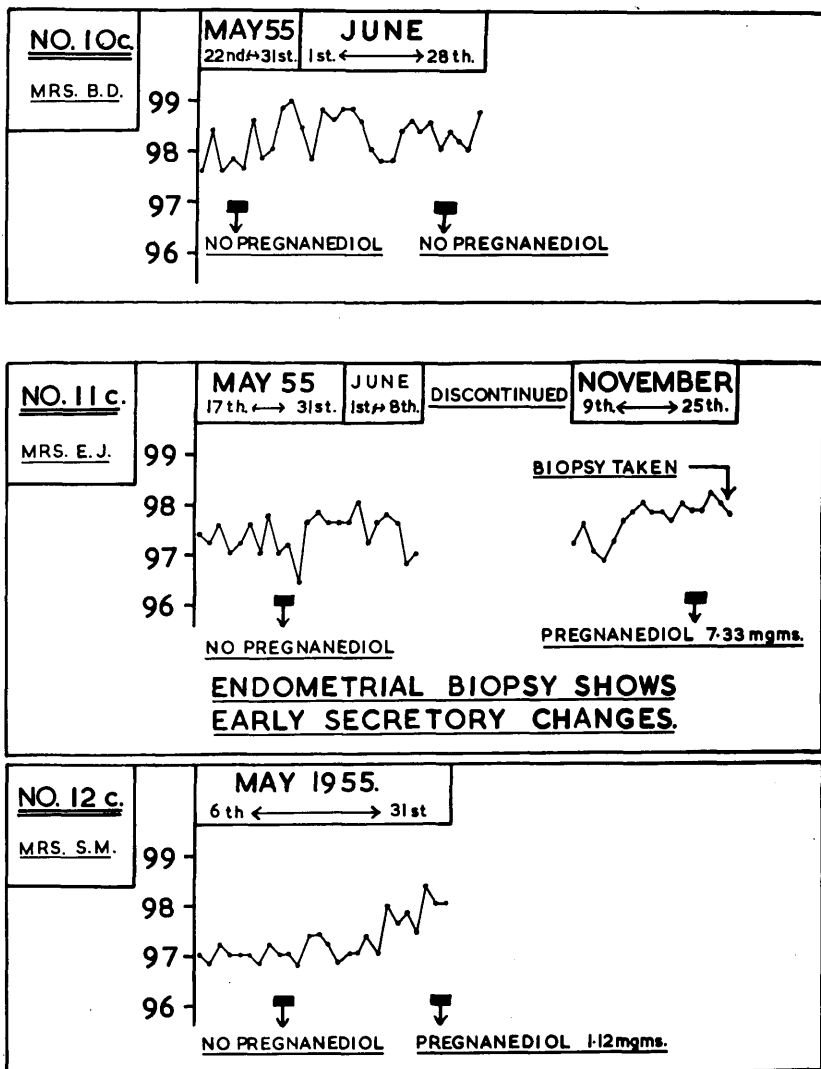


Fig. XX showing the temperature records of subjects 10c, 11c and 12c with the results of pregnanediol estimations and endometrial biopsies.



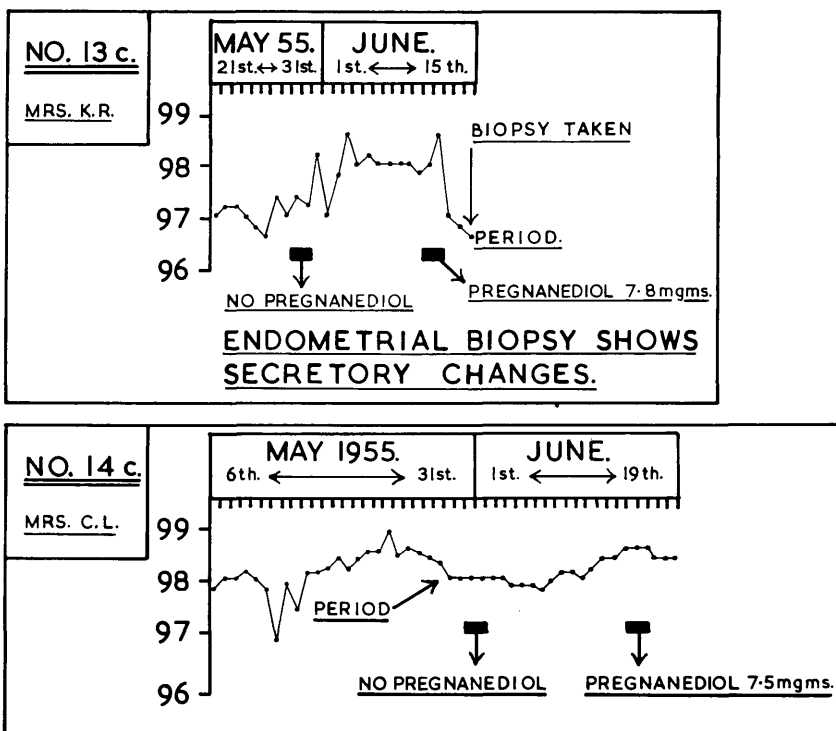


Fig. XXI showing the temperature record of subjects 13c and 14c with the results of pregnanediol estimations and endometrial biopsies.

Table XV

A Classification of the Temperature Records of the Subjects of Group (a), i.e. those who had undergone the operation of Hysterectomy with Conservation of One or Both Ovaries.

No.	Initials	Approx. Age at time of Investi- gation	Total or Subtotal Operation	Interval since Operation	Pattern of Temperature Record - Diphasic or Monophasic
<u>Cases of Hysterectomy with Conservation of Both Ovaries</u>					
1a	Mrs. B.D.	40 6/12ths	T	3	Diphasic
2a	" M.K.	42 8/12ths	T	1 5/12ths	Do.
3a	" J.F.(D)	49 4/12ths	T	2 5/12ths	Monophasic
4a	" E.D.	46 9/12ths	T	1 6/12ths	Do.
5a	" J.F.(B)	46 2/12ths	T	1 10/12ths	Do.
6a	" J.C.(K)	42 9/12ths	T	2 10/12ths	Prob.Diphasic
7a	" M.McD	43 6/12ths	S	3	Diphasic
8a	" J.C.(B)	36 8/12ths	T	2 6/12ths	Do.
9a	" E.H.(C)	37 1/12th	T	2 2/12ths	Do.
10a	" H.T.	40 9/12ths	T	2	Do.
11a	" C.K.	39	S	1 7/12ths	Prob.Diphasic
12a	" A.R.	47 3/12ths	T	2 5/12ths	Diphasic
13a	" M.H.(T)	41 1/12th	S	6 6/12ths	Do.
14a	" J.S.	39 6/12ths	T	1 10/12ths	Monophasic
15a	" I.R.	45 1/12th	S	1 6/12ths	Diphasic
16a	" J.McI	48 8/12ths	T	1 8/12ths	Prob.Diphasic
<u>Cases of Hysterectomy with Conservation of One Ovary</u>					
17a	" M.W.	33 6/12ths	S	1 5/12ths	Diphasic
18a	" H.P.	46	T	1 7/12ths	Do.
19a	" E.C.	46 7/12ths	S	2 5/12ths	Do.
20a	" J.O'N	40 4/12ths	S	1 4/12ths	Prob.Diphasic
21a	" M.D.	48 8/12ths	T	1 7/12ths	Do.
22a	" J.B.	40 9/12ths	T	5 6/12ths	Monophasic
23a	" M.S.	51	T	1 10/12ths	Do.
24a	" M.C.	41 9/12ths	S	7	Do.

As the records extend over several months, it is obviously impossible to express the age of the patient with the precision of the other investigations. The figure given here therefore represents the subject's approximate age midway through the period of her record-keeping.

Table XVI

A Comparison of the Patterns of the Temperature Records of the Subjects of group (c) (i.e. normal women) with the Histological Appearances of the Endometrium during the second half of the same menstrual cycle.

<u>No.</u>	<u>Initials</u>	<u>Age</u> <u>Yrs.</u>	<u>Type of</u> <u>Temperature</u> <u>Record</u>	<u>Histological Appearance</u> <u>of Endometrial Biopsy</u> <u>taken during second half</u> <u>of menstrual cycle.</u>
1c	Mrs. W.D.	40 1/12th	Diphasic	No biopsy obtained.
2c	" S.D.	43 4/12ths	Monophasic	Secretory changes
3c	" M.R.	43	Indeterminate	Do.
4c	" D.R.	38 6/12ths	Monophasic	Do.
5c	" J.K.	41 5/12ths	Diphasic	Few glands show subnuclear vacuolation.
6c	" J.H.	40	Do.	Secretory changes
7c	" C.W.	48 7/12ths	Monophasic	Proliferative endometrium
8c	" N.McG	49 2/12ths	Do.	Do.
9c	" G.S.	39 6/12ths	Do.	Secretory changes
10c	" B.D.	42 6/12ths	Do.	No biopsy obtained
11c	" E.J.	43 11/12ths	Diphasic	Secretory changes
12c	" S.M.	42 8/12ths	Do.	Proliferative endometrium
13c	" K.R.	42 3/12ths	Do.	Secretory changes
14c	" C.L.	42 1/12th	Do.	No biopsy obtained.

Table No.XVII

A Classification of the Temperature Records of those subjects of group (a) whose ovaries were both conserved at hysterectomy.

Number Exhibiting Diphasic Temperature Records	= 9	} (75%)
Number " Records which were probably Diphasic	= 3	
Number Exhibiting Monophasic Temperature Records	= 4	(25%)
Total	= 16	

Table No.XVIII

A Classification of the Temperature Records of those subjects of group (a) who had one ovary conserved at hysterectomy.

Number exhibiting Diphasic Temperature Records	= 3	} (62.5%)
" " Records which were probably Diphasic	= 2	
Number exhibiting Monophasic Temperature Records	= 3	(37.5%)

Table No.XIX

A Classification of the Temperature Records of those subjects of group (a) who had undergone the operation of subtotal (i.e. supravaginal) hysterectomy

Number exhibiting Diphasic Temperature Records	= 5	} (87.5%)
" " Records which were probably Diphasic	= 2	

Number exhibiting Monophasic Temperature Records	= 1 (12.5%)
Total	= 8

Table No. XX

A Classification of the Temperature Records of those subjects of group (a) who had undergone the operation of total hysterectomy

Number exhibiting Diphasic Temperature Records	= 7	} (62.5%)
" " " Records which were probably Diphasic	= 3	
Number exhibiting Monophasic Temperature Records	= 6 (37.5%)	
Total	= 16	

D I S C U S S I O N

It was not considered necessary to reproduce, in this work, the complete temperature record of each subject, though a characteristic portion has, in every instance, been presented as a means of illustrating not only the results of this investigation, but also those of the one which follows it.

As was to be expected, those subjects whose ovaries had been removed/

removed when hysterectomy was performed, returned temperature records lacking any discernible rhythmic variation. In many, the readings fluctuated within narrow limits except when an acute illness relieved the monotony of the pattern (See Figs. IX to XVI).

The basal temperature readings of the normal subjects (i.e. those of group (c)), were in most cases recorded throughout one menstrual cycle only, and it is significant that in only half was the diphasic pattern of the ovulating woman clearly detectable (see Fig.XVII to XXI). Ten of the fourteen subjects of this group agreed to allow an endometrial biopsy to be taken during the second half of the cycle, and while there was no instance in which the evidence of a diphasic temperature record was contradicted by the histological appearance of the endometrium, it is noteworthy that four subjects (Nos. 2c, 3c, 4c and 9c) whose temperature records could not possibly be described as typically diphasic in character, nevertheless exhibited presumptive evidence of ovulation, in that endometrial biopsies from these women all showed secretory changes.

In those cases in which the endometrial biopsy showed only proliferative changes during the second half of the cycle, the/

the temperature record was found to be monophasic (Nos. 7c, and 8c).

It would appear, therefore, that while the temperature record failed in some instances to indicate that ovulation had occurred, in no case did it mislead one into suspecting ovulation when endometrial appearances indicated that this had not occurred.

The discrepancy between temperature chart and endometrial biopsy was probably mainly due to the subjects' lack of experience in keeping temperature records, though it may also indicate the limitations of the temperature chart as an indicator of corpus luteum formation.

Great pains were taken over the temperature records of the main investigation group, i.e. the subjects of group (a), and months were spent in attempting to instruct these women in the use of the thermometer. Those portions of the records which have been reproduced here are the ones which were thought to be most typical of the general trend. In the records of some of the subjects of this group, the recurrence of the diphasic pattern could be observed month after month, while in others a monophasic record persisted during the entire period of the investigation.

An/

An intermediate group, in which an occasional sustained elevation of temperature hinted at a possible "thermal shift", was less easy to classify. In Table XV which differentiates the records as "monophasic" or "diphasic", the word "probable" has been inserted when interpretation was particularly difficult, and this has been followed by whichever of these two words seemed the more applicable as a term descriptive of that particular record.

Twelve (50%) of the subjects of this group were considered to exhibit temperature records which could, without hesitation, be described as diphasic, while seven (29.17%) were equally definitely monophasic. The remaining five (20.83%) have been designated as "probably diphasic".

The results of this investigation strongly suggest that, in at least 50% of those subjects whose ovaries had been conserved at hysterectomy, there was evidence of continuing ovulation. The fact that the diphasic pattern occurred regularly in cyclical fashion in 12 of the 24 subjects of this group virtually precludes any alternative explanation.

Even among those in whom the two phases were less easily distinguished there remained the possibility that corpus luteum formation might be occurring, if not in every cycle, at least, sporadically, though in these women it must be conceded that the evidence/



evidence for ovulation is not by any means conclusive.

The fact that seven women of this group returned records which remained consistently monophasic for the period during which daily temperatures were taken, suggests that ovulation probably did not then occur. It does not, of course, argue a complete cessation of ovarian activity, as anovular cycles are not infrequently found in those years immediately preceding the menopause (Sharman 1955).

Although the evidence in favour of continuing ovulation was stronger among those women whose ovaries were both conserved as compared with those who had only one ovary, the results do not manifest a difference sufficiently striking to justify one drawing any conclusions on this score, added to which is the fact that the series is too small a one for the difference to be significant.

Since several writers (Tamis 1934, Marx, Catchpole and McKennon 1936) contend that the supravaginal hysterectomy is followed by a less severe menopause than the more radical operation, it is interesting to observe that in 87.5% of those who underwent the subtotal operation with conservation of one or both ovaries, the temperature record indicated a probable continuance of ovulation whereas, among the larger group upon whom total hysterectomy had been performed 62.5% appeared to ovulate.

In/

In a relatively small series like this one, the difference between these two sets of figures is, of course, without real significance.

The important fact which does emerge is that a certain number, both of those women who had undergone total hysterectomy and of those others who had undergone subtotal hysterectomy, returned temperature records which seemed to suggest that ovulation may continue years after either operation has been performed.

One very interesting feature of this investigation was the fact that Mrs. M.H.(T)(No.13a) returned a temperature record in which the diphasic pattern recurred again and again. Not only so, but as has already been stated, this patient whose hysterectomy had been of the supravaginal type, continued to "menstruate" (for one to two days) six and a half years after the greater part of her uterus had been removed. It is worthy of note that this woman was one of the five of group (a) who complained of what she, at least, believed to be "hot flushes".

Of the remaining four women of this group who complained of "hot flushes", three (Nos. 22a, 23a and 24a) returned temperature records which were definitely monophasic, but one (No. 21a) submitted a chart which bore more than a slight resemblance to the classical pattern of the ovulating subject and was therefore classified as "probably diphasic".

SUMMARY and CONCLUSIONS

This investigation is based upon the fact that the basal temperature record of a regularly ovulating woman exhibits a characteristic pattern. During the oestrogenic phase of the cycle the temperature will be found to show only minor variations, and will be relatively low, but about the time when ovulation occurs it rises by about .6 to 1° F, and thereafter maintains this relatively higher level, until just before the next menstrual period, when something like a pre-ovulation reading is again obtained.

This recurrent rise in temperature is known as the "thermal shift" and is believed to be due to the liberation of progesterone by the corpus luteum.

The temperature records of girls up to the time of puberty, like those of postmenopausal women, oophorectomised women and men, do not exhibit this diphasic pattern, which however may be artificially produced by the injection of progesterone.

The naturally occurring diphasic temperature record is consequently accepted as evidence of ovulation which precedes corpus luteum formation.

A brief account is given of how this phenomenon came to be observed and explained.

Davis and Fugo (1948) noted what they believed to be the "thermal shift" and diphasic pattern in the temperature records of 4 out of 12 patients during the few months following hysterectomy with conservation of ovaries.

This publication is briefly discussed.

No other instance was found in the literature of the basal temperature record having been used to ascertain whether or not ovulation was occurring in hysterectomised subjects.

In this investigation each subject was asked to keep a daily record of her basal morning temperature over a period which ranged from several weeks to five months.

As was to be expected, all subjects who had undergone the operation of hysterectomy with bilateral oophorectomy, returned temperature records which were monophasic in character.

Half of the "normal" subjects (who had undergone no operation) exhibited the diphasic pattern, although some of those whose temperature records appeared to be monophasic, were found, during the same cycle, to have an endometrium which/

which showed secretory changes. The discrepancy between temperature record and endometrial histology is thought to be due to the inexperience of this group of women in keeping temperature records. Unlike those women of groups (a) and (b), they did not record their temperatures daily for several months.

Fifty per cent of the women of group (a), i.e. those whose uterus had been removed but who still retained one or both ovaries, returned temperature records which exhibited the diphasic pattern characteristic of the ovulating subject. In another 20.85% the evidence, though less convincing, pointed to occasional, though not regularly recurring, ovulation. The records of 29.17% of the women of this group were clearly monophasic, and gave no hint that ovulation had occurred during the period when temperature records were being kept.

Some of the women of group (a) who had undergone total, and some others who had undergone subtotal, hysterectomy submitted temperature records showing the diphasic pattern, as did a number of the women who had one ovary and some of those who had both ovaries, conserved when hysterectomy was performed.

The/

The general conclusion to be drawn from this investigation is that about half of the subjects of group (a) showed strong evidence of continuing ovulation, and that ovulation may also, on the evidence of the basal temperature record, have occurred occasionally in another 20.83% of the subjects of this group. The temperature records of 29.17% of the subjects of this group showed no evidence of ovulation.

Continued ovulation would therefore appear to be consistent with the subject having previously undergone either total or subtotal hysterectomy with conservation of one or both ovaries.

## SECOND INVESTIGATION

The object of the investigation was to determine the  
effect of the presence of the gas on the rate of  
oxidation of the metal. The results of the  
investigation are given in the following table.  
The temperature of the metal was kept constant  
at 100°C. The rate of oxidation was measured  
by the weight loss of the metal. The results  
show that the rate of oxidation is increased  
by the presence of the gas. The rate of  
oxidation is also increased by the presence  
of the gas when the metal is heated to  
150°C. The rate of oxidation is also  
increased by the presence of the gas when  
the metal is heated to 200°C. The rate  
of oxidation is also increased by the  
presence of the gas when the metal is  
heated to 250°C. The rate of oxidation  
is also increased by the presence of the  
gas when the metal is heated to 300°C.

## THIRD INVESTIGATION

The object of the investigation was to determine the  
effect of the presence of the gas on the rate of  
oxidation of the metal. The results of the  
investigation are given in the following table.  
The temperature of the metal was kept constant  
at 100°C. The rate of oxidation was measured  
by the weight loss of the metal. The results  
show that the rate of oxidation is increased  
by the presence of the gas. The rate of  
oxidation is also increased by the presence  
of the gas when the metal is heated to  
150°C. The rate of oxidation is also  
increased by the presence of the gas when  
the metal is heated to 200°C. The rate  
of oxidation is also increased by the  
presence of the gas when the metal is  
heated to 250°C. The rate of oxidation  
is also increased by the presence of the  
gas when the metal is heated to 300°C.

I N T R O D U C T I O N

The object of this investigation was to revalue the tentative conclusions of the previous investigation by the application of an additional test of their validity.

Among those temperature records not obviously monophasic there was wide variation. The curve in some bore a recognisable resemblance to the ovulatory cycle as it has often been described in gynaecological literature, while, in other cases, the likeness was less pronounced. Where there was the suspicion of a "thermal shift" but no more than a suspicion, the record was difficult to classify and its significance remained doubtful.

It was felt that if the hint of a "thermal shift" in a woman's temperature record and the subsequent suggestion of a high temperature phase could be supported by other evidence of the production of progesterone on the part of the ovary, the probability that ovulation had occurred in that particular subject would be enhanced.

From urinary pregnanediol estimations can be derived precisely the type of confirmation which is required, and the third investigation consisted in having the urine of the various/



various subjects of each group estimated for pregnanediol at times selected in accordance with information supplied by the temperature records.

### Pregnanediol a Metabolite of Progesterone

Pregnanediol was first isolated by Marrian in 1929 and shortly afterwards its molecular structure was described by Butenandt (1930).

Its chemical affinities with progesterone immediately led to the supposition that it might be a metabolite of that hormone. This probability gained in strength with reports that Venning and Browne (1937) had recovered sodium pregnanediol glucuronidate from the urine of normal women of childbearing age, and that relatively higher values were obtained by Browne, Henry and Venning from the urine of pregnant women (1937). This, of course, is in keeping with the postulated relationship between the two substances since, in pregnancy, both the corpus luteum and the placenta are engaged in the production of progesterone.

The inability of Buxton and Westphal (1939) to detect any pregnanediol in the urine of men unless they had previously been injected/

injected with progesterone seemed also to agree with the theory that pregnanediol was a metabolite of progesterone, though it should be added that a more recent and more sensitive method of pregnanediol estimation has disclosed that pregnanediol is to be found in the urine of the human male as a normal constituent (Klopper 1955).

Pregnanediol is not the only metabolite of progesterone, for the amount of this substance excreted in the urine of experimental subjects has been shown to account for only a small proportion of the progesterone injected by the intramuscular route (de Watteville 1951).

It is also true that pregnanediol may be a metabolite of substances other than progesterone, for example, desoxycorticosterone (Cuyler 1940).

Hyperactivity of the adrenal has been observed to be associated with an increased urinary output of pregnanediol (Salmon 1941).

The broad generalisation that urinary pregnanediol is mainly derived from progesterone may, however, still be considered sound, since that fraction which comes from extra-ovarian sources is relatively small in the normal individual, and many of the standard clinical methods in use for its estimation in the urine frequently fail to detect its presence in a 24-hour sample.

Pregnanediol and the Menstrual Cycle

If the pregnanediol which is found in the urine of a normal woman is to be regarded as predominantly derived from endogenous progesterone, one would expect relatively small amounts of it to be present in 24-hour samples of urine collected during the proliferative phase of the menstrual cycle. Venning and Browne (1940) using the Venning method of sodium pregnanediol glucuronidate estimation, conducted such an investigation and reported consistently negative results. The observation was confirmed by de Watteville (1951), who, employing a different method of pregnanediol estimation, failed to detect this substance in the urine of normal women during this phase of the cycle.

As progesterone is the hormone of the corpus luteum, (and of the placenta), one would expect that 24-hour samples of urine collected during the postovulatory phase of the cycle should, however, contain appreciable amounts of pregnanediol. While this was found by Rogers and Sturges (1950) to be very frequently true, they also reported that in their series, estimations conducted on some 24-hour samples of postovulatory urine yielded no pregnanediol.

Rogers/

Rogers and Sturges (1950) in their estimations of "free" pregnanediol, discovered that its appearance in the urine might precede, coincide with, or follow, that rise in temperature which is believed to signify the liberation of progesterone by the corpus luteum, and have suggested that its occasional discovery in the urine prior to the "thermal shift" might be due to its derivation from progesterone produced by "luteinised theca prior to ovulation". Davis and Fugo (1948) who detected small amounts during this phase of the cycle attributed them to extraovarian sources.

#### Progesterone and the Uterus

When Browne and Venning (1938) failed to recover sodium pregnanediol glucuronidate from the urine of two hysterectomised subjects following injection of 24 mg. of progesterone, it was thought by them that the uterus probably played a necessary part in the metabolism of progesterone, a theory which seemed to be supported by the report that Hamblin and his co-workers (1939) were unable to detect sodium pregnanediol glucuronidate in the urine of a few subjects who had undergone endometrial curettage.

Buxton/

Buxton and Westphal (1939), however, by demonstrating that sodium pregnanediol glucuronidate was to be found in the urine of men who had been given injections of progesterone effectively disposed of this belief which is no longer held.

#### PREVIOUS INVESTIGATIONS

A thorough search of the literature disclosed only two attempts to assess ovarian function following hysterectomy by means of pregnanediol estimations of the urine.

Jones and Te Linde (1941) conducted a number of urinary pregnanediol estimations on three women who had undergone the operation of hysterectomy six months previously, and succeeded in obtaining 3.3 mgm. of sodium pregnanediol glucuronidate from the urine of one of them. Tests on the urine of the other two yielded only negative results.

The other investigation was by Davis and Fugo (1948), who conducted estimations of sodium pregnanediol glucuronidate on the urine of twelve women who had undergone hysterectomy within the previous four months. They record that from the urine of some of their subjects they recovered amounts of pregnanediol ranging from 5 to 17 mgms. per 24-hour sample/

sample during what, by temperature record, they assumed to be the postovulatory phase of the cycle.

#### M E T H O D

In this investigation all three groups of women were tested.

Those whose ovaries had been removed, i.e. group (b), each submitted one 48-hour sample of urine, which was submitted for pregnanediol estimation.

From the normal subjects, i.e. those of group (c), two 48-hour samples were taken, one in the earlier part of the cycle at a time when the temperature record suggested that the "thermal shift" had not yet occurred. The second sample was taken during the latter half of the cycle irrespective of whether or not there was evidence of a significant elevation of temperature. Both were estimated quantitatively for pregnanediol.

The results obtained from each of the above groups were used as controls, the main subjects of investigation being those of group (a), i.e. those subjects from whom the uterus had been removed but who still retained one or both ovaries.

From/

From each of these women two 48-hour samples of urine were obtained and submitted for pregnanediol estimation. The object was to submit, whenever possible, one sample which had been collected during the low-temperature phase of the cycle and one which had been collected during the high-temperature phase. In some cases the temperature record was such that it was not difficult to differentiate the two phases, but as has already been explained, certain records did not conform very closely to the typical diphasic pattern. In such instances one of the samples was collected when the temperature had been elevated for a few days, though, as the records will reveal, the elevation was sometimes of short duration. The other was collected when the temperature was relatively lower.

Where there was no indication whatsoever of a diphasic pattern, two 48-hour samples were collected more or less at random, but with an interval of at least two weeks between one and the other.

The estimations were performed at the Fife District Laboratory.

As only a very limited number of these estimations could be undertaken each week, it was not always possible to submit

a 48-hour sample on the first occasion on which the temperature record led one to believe that corpus luteum formation had probably recently occurred.

For the same reason, a gap of several months sometimes elapsed between the performance of the two pregnanediol estimations of the same subject.

Usually but, of course not invariably, the first sample was submitted after the temperature record showed a significant and sustained rise, so that, in most instances, the "high temperature" sample of urine was estimated before the "low temperature" sample.

#### Method of Pregnanediol Estimation

The method of pregnanediol estimation adopted in this investigation was that described by de Watteville, Borth and Gsell (1948). Although the writer makes no claim to any specialised knowledge of biochemistry, it is appropriate that the procedure adopted in performing these estimations should be described in some detail. The following account of the method is based on information supplied by Dr. Easson who supervised the performance of the tests.



Technique.

Except where high values are expected, as in pregnancy after forty days, one litre of urine and 50 ml. of redistilled toluene are heated to boiling. One hundred ml. of concentrated hydrochloric acid are added slowly and the mixture is refluxed for 15 minutes, and then cooled rapidly under the tap.

The clear layer of urine is then drawn off and re-extracted twice with 25 ml. portions of toluene using gentle shaking. The combined toluene extracts and emulsions are next washed with 2 normal sodium hydroxide in 20% sodium chloride using two to four 25 ml. portions, until the washings remain clear and the emulsion is broken down. The sodium hydroxide is removed by two final washings with 25 ml. portions of distilled water.

If high values are expected, 100 ml. of urine, 50 ml. of toluene and 10 ml. of hydrochloric acid are refluxed for fifteen minutes, followed by careful but thorough shaking and rapid cooling under the tap. The toluene layer and the emulsions are combined and washed with two 15 ml. portions of 2 normal sodium hydroxide in 20% hydrochloric acid solution and twice with 15 ml. of distilled water.

The toluene extract obtained from 100 or 1,000 ml. of urine is heated on a hot-plate until the last traces of water are dispelled. The solution is then allowed to cool down to 50-60° C, and then 10 ml. of 2% methyl alcoholic sodium hydroxide are added and the mixture is reduced to 20-25 ml. The toluene solution is allowed to cool and is filtered through a filter of medium porosity by suction. The precipitate is washed with three successive small amounts of hot toluene. The filtrate is concentrated on the hot-plate to 4-6 ml. and allowed to cool.

The toluene extract is allowed to percolate through the chromatographic column and the column washed with 6 ml. of benzene, all residues being preserved for use in the event of a high result being found. The pregnanediol is then eluted with 10 ml. of 10:1 benzene-absolute alcohol mixture, the eluate being collected into a weighted round-bottomed flask. The residue obtained after evaporation to complete dryness is washed in .5 to 1 ml. of cold ether to remove coloured impurities and the remaining white crystalline pregnanediol is dried by evaporation in vacuo and in a dessicator and finally weighed.

If/

If more than 6 to 15 mg. of pregnanediol are expected a larger column is used. If 6 mg. or more are found with the normal column, the toluene and benzene eluates should be concentrated and passed through a second column.

#### Apparatus

The chromatographic column is 7-9 mm. in diameter and 10-15 mm. high, and is prepared by sedimentation of 200-300 mg. of aluminium oxide in dry benzene. Pressure is not generally necessary for percolation of the toluene extracts, but may be employed if required. The column will absorb 6-15 mg. of pregnanediol.

#### Reagents

The aluminium oxide recommended is Merck's 'Activity 1' according to Brockmann'. Other preparations may be tested for suitability by recovery experiments with normal male urine to which pregnanediol has been added. Recovery is 95-100%.

The toluene is pure 'redistilled' reagent.

The sodium hydroxide-sodium chloride solution for washing is made by dissolving 200 gm. of sodium chloride and 80 g. of sodium hydroxide in water and making up to 1,000 ml. It should be removed by pipette in order to avoid disturbing any sediment. The 2% (0.5M) sodium hydroxide in methyl alcohol is prepared by dissolving 5 g. of the solid in 250 ml. of pure methanol (A.R. grade), preferably by use of a mechanical shaker. It is filtered and stored in a bottle with a well-fitting glass stopper. It should be replaced if it becomes cloudy or coloured.

RESULTS

Table XXI

Showing results of urinary pregnanediol determinations of subjects of group (a) (i.e. cases of hysterectomy with conservation of one or both ovaries) in relation to Basal Temperature Record

<u>No.</u>	<u>Initials</u>	<u>Approx. Age</u> <u>at time of</u> <u>Investi-</u> <u>gation</u> <u>Years</u>	<u>Approx.</u> <u>Inter-</u> <u>val</u> <u>since</u> <u>Hyst.</u> <u>Years</u>	<u>Type</u> <u>of</u> <u>Hyst.</u>	<u>Patt.</u> <u>of</u> <u>Tem-</u> <u>pera-</u> <u>ture</u> <u>Record</u>	<u>Amount</u> <u>of Preg-</u> <u>nanediol</u> <u>in 48-hr.</u> <u>sample</u> <u>of urine</u> <u>(low tem-</u> <u>perature</u> <u>phase of</u> <u>cycle</u> <u>if di-</u> <u>phasic</u>	<u>Amount</u> <u>of Preg-</u> <u>nanediol</u> <u>in 48-hr.</u> <u>sample</u> <u>of urine</u> <u>(high</u> <u>tempera-</u> <u>ture</u> <u>phase of</u> <u>cycle</u> <u>if di-</u> <u>phasic</u>	
<u>Cases with Conservation of Both Ovaries</u>								
1a	Mrs. B.D.	40	6/12ths	3	Total	Diphasic	.26 mgm.	14.75 mgm.
2a	" M.M.	42	8/12ths	1	"	"	Nil	12.75 "
3a	" J.D.(D)	49	4/12ths	2	"	Monophasic	Nil	Nil
4a	" E.D.	46	9/12ths	1	"	"	"	"
5a	" J.F.(B)	46	2/12ths	1	"	"	"	"
6a	" J.C.(K)	42	9/12ths	2	"	Prob. Di-	.8 mgm.	2.08 mgm.
7a	" M.McD	43	6/12ths	3	Sub-	phasic		
					total	Diphasic	Nil	5.7 "
8a	" J.C.(B)	36	8/12ths	2	Total	"	"	6.2 "
9a	" M.H.(C)	37	1/12th	2	"	"	"	7.6 "
10a	" H.T.	40	9/12ths	2	"	"	"	.9 "
11a	" C.K.	39	7/12ths	1	Sub-	Prob. Di-	.28 mgm.	.66 "
					total	phasic		
12a	" A.R.	47	3/12ths	2	Total	Diphasic	"	1 "
13a	" M.H.(T)	41	1/12th	6	Sub-	"	.15 mgm.	3.78 "
					total			
14a	" J.S.	39	6/12ths	1	Total	Monophasic	Nil	Nil
15a	" I.R.	45	1/12th	1	Sub-total	Diphasic	Nil	Nil
16a	" J.McI	48	8/12ths	1	Total	Prob. Di-	Nil	.6 mgm.
						phasic		

<u>No.</u>	<u>Initials</u>	<u>Approx. Age</u> <u>at time of</u> <u>Investi-</u> <u>gation.</u> <u>Years</u>	<u>Approx.</u> <u>Inter-</u> <u>val</u> <u>since</u> <u>Hyst.</u> <u>Years</u>	<u>Type</u> <u>of</u> <u>Hyst.</u>	<u>Patt.</u> <u>of</u> <u>Tem-</u> <u>pera-</u> <u>ture</u> <u>Record</u>	<u>Amount</u> <u>of Preg-</u> <u>nandiol</u> <u>in 48-hr.</u> <u>sample</u> <u>of urine</u>	<u>Amount</u> <u>of Preg-</u> <u>nandiol</u> <u>in 48-hr.</u> <u>sample</u> <u>of urine</u> <u>(high</u> <u>tempera-</u> <u>ture</u> <u>phase of</u> <u>cycle</u> <u>if di-</u> <u>phasic</u>
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Cases with Conservation of One Ovary

17a	Mrs. M.W.	33	6/12ths	1	5/12ths	Sub- total	Diphasic	.15 mgm.	4.2 mgm.
18a	" H.P.	46		1	7/12ths	Total	"	Nil	.65 "
19a	" E.C.	46	7/12ths	2	5/12ths	Sub- total	"	"	.9 "
20a	" J.O'N	40	4/12ths	1	4/12ths	Do.	Prob. Di- phasic	"	.84 "
21a	" M.D.	48	8/12ths	1	7/12ths	Total	Do.	"	3.8 "
22a	" J.B.	40	9/12ths	5	6/12ths	Do.	Monophasic	"	Nil
23a	Miss M.S.	51		1	10/12ths	Do.	Do.	"	"
24a	Mrs. M.C.	41	9/12ths	7		Sub- total	Do.	"	"

Table XXII

Showing Results of Urinary Pregnanediol Estimations of Subjects of Group (b) (i.e. women who had undergone hysterectomy with bilateral oophorectomy).

<u>No.</u>	<u>Initials</u>	<u>Age at Time of Investi- gation Years</u>	<u>Interval since Hyster- ectomy Years</u>	<u>Type of Hyster- ectomy</u>	<u>Amount of Pregnanediol in 48-hour sample of urine</u>
1b	Mrs. A.S.	42 2/12ths	1 9/12ths	Total	Nil
2b	" A.Y.	45	2 1/12th	"	"
3b	" A.C.	45 11/12ths	2 5/12ths	"	"
4b	" M.W.(C)	39 9/12ths	9 4/12ths	Subtotal	"
5b	" H.W.	48 6/12ths	3	Total	"
6b	Miss B.F.	49 4/12ths	1 8/12ths	"	"
7b	Mrs. M.B.	42 6/12ths	1 7/12ths	"	"
8b	" J.B.	49 6/12ths	2 11/12ths	"	"
9b	" M.S.(C)	51 9/12ths	2 3/12ths	"	"
10b	" A.H.	39 11/12ths	1	"	"
11b	" M.S.(K)	44 11/12ths	1 9/12ths	"	"
12b	" C.O'G	41 5/12ths	2 7/12ths	"	"
13b	" M.P.	42 6/12ths	3 4/12ths	Subtotal	"
14b	" P.F.	30 6/12ths	1 6/12ths	Total	"
15b	" A.T.	33 8/12ths	1 11/12ths	Subtotal	"
16b	" M.D.	42 8/12ths	2 4/12ths	Do.	"
17b	" C.P.	48 5/12ths	1 8/12ths	Do.	"
18b	" M.W.(D)	43	1 1/12th	Total	.25 mgn.
19b	" E.M.	45 5/12ths	1 5/12ths	Subtotal	Nil
20b	" A.W.	47 1/12th	2 1/12th	Total	"
21b	Miss J.McG	43 10/12ths	2 3/12ths	"	"
22b	Mrs. A.E.	49 10/12ths	1 9/12ths	Subtotal	"
23b	" E.Y.	51 6/12ths	2 5/12ths	Total	"
24b	" E.H.	46 10/12ths	1 10/12ths	"	"

Table XXIII

Showing Results of Urinary Pregnanediol determinations of subjects of group (c) (i.e. women who had undergone neither hysterectomy nor oophorectomy).

<u>No.</u>	<u>Initials</u>	<u>Age at Time of Investigation, Years</u>	<u>Pattern of Temperature Record</u>	<u>Amount of Pregnanediol in 48-hr. sample of urine (1st half of menstrual cycle)</u>	<u>Amount of Pregnanediol in 48-hr. sample of urine (2nd half of menstrual cycle)</u>	<u>Histological Appearance of Endometrium in 2nd half of menstrual cycle</u>
1c	Mrs. W.D.	40 1/12th	Diphasic	Nil	6.33 mgms.	No biopsy
2c	" S.D.	43 4/12ths	Monophasic	Nil	6.1 "	secretory changes
3c	" M.R.	43	Indeterminate	Nil	.9 "	Do.
4c	" D.R.	38 6/12ths	Monophasic	Nil	1.4 "	Do.
5c	" J.K.	41 5/12ths	Diphasic	Nil	7.5 "	Subnuclear vacuolation
6c	" J.H.	40	Do.	.8 mgms.	2.6 "	secretory changes
7c	" C.W.	48 7/12ths	Monophasic	Nil	Nil	Proliferative endometrium
8c	" N.McG	49 2/12ths	Do.	Nil	Nil	Do.
9c	" G.S.	39 6/12ths	Do.	Nil	5.9 "	Secretory changes
10c	" B.D.	42 6/12ths	Do.	Nil	Nil	No biopsy
11c	" E.J.	43 11/12ths	Diphasic	Nil	7.33 "	Secretory changes
12c	" S.M.	42 8/12ths	Diphasic	Nil	1.2 "	No biopsy.
13c	" K.R.	42 3/12ths	Diphasic	Nil	7.8 "	Secretory changes
14c	" C.L.	42 1/12th	Diphasic	Nil	7.55 "	No biopsy

FIG. XXII.

DIAGRAMATIC REPRESENTATION OF THE RESULTS OF PREGNANEDIOL ESTIMATIONS OF TWO 48 HOUR SAMPLES OF URINE OBTAINED FROM EACH OF THE SUBJECTS OF GROUP (a) Where the patients' temperature record was diphasic the first value shown is that obtained during the "low temperature phase" of the cycle, the second that obtained during the "high temperature phase" of the cycle. Where the record was monophasic the two samples were obtained at times separated by an interval of at least two weeks.

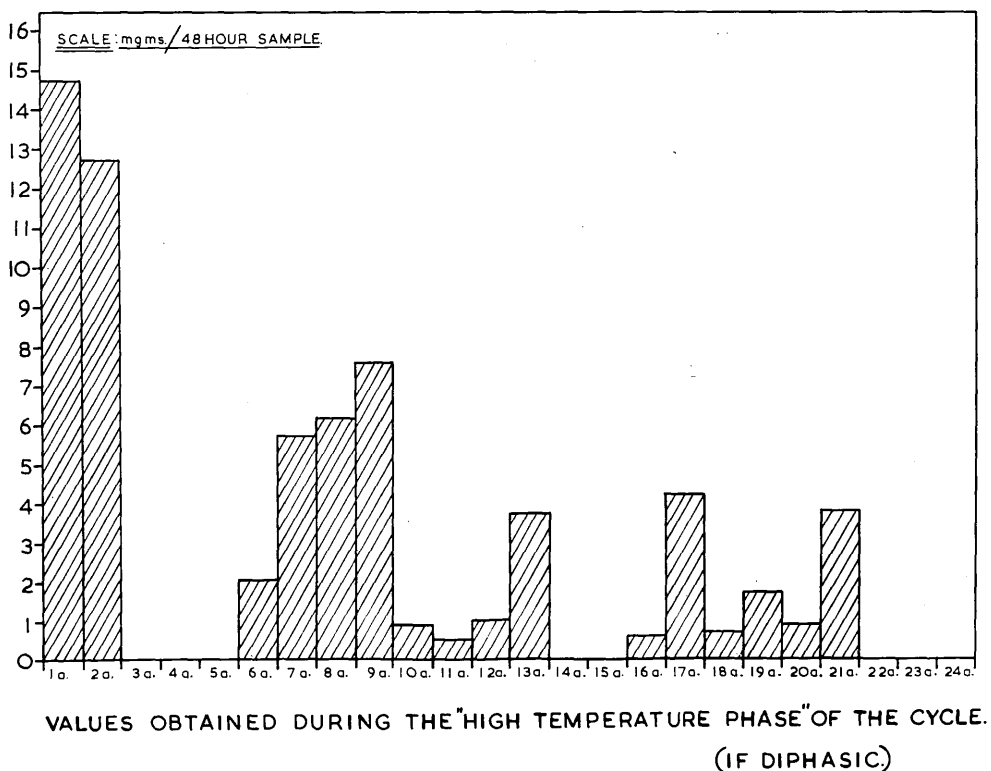
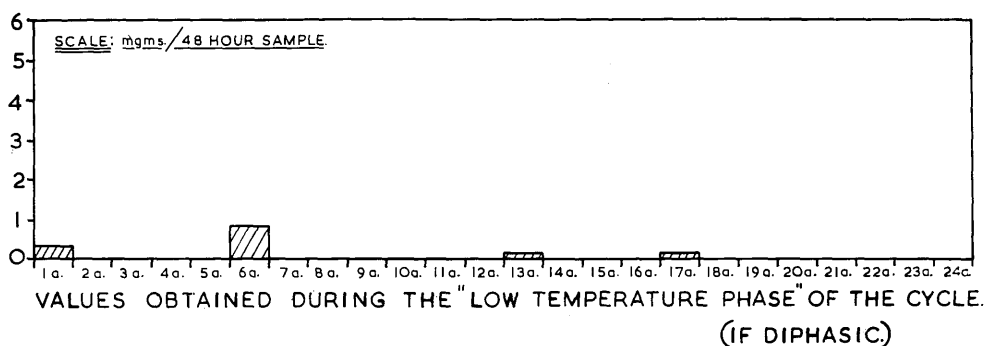
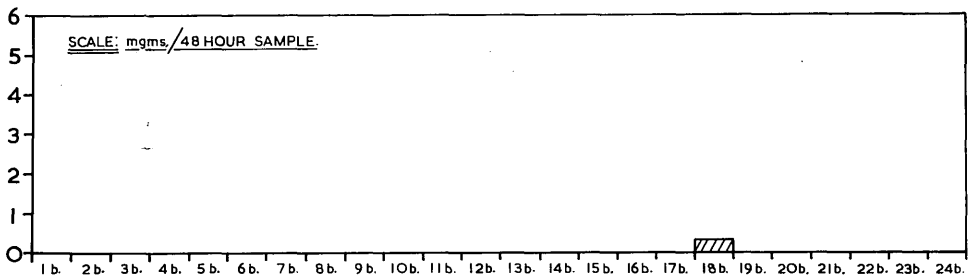


FIG. XXIII.

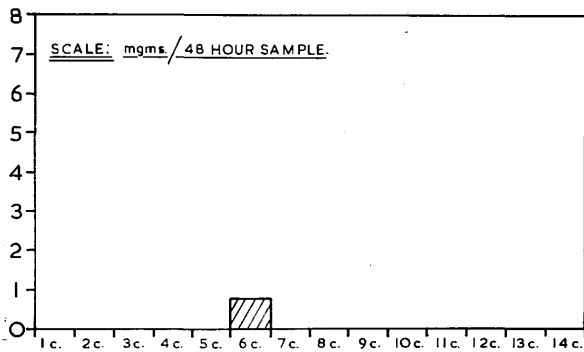
DIAGRAMATIC REPRESENTATION OF THE RESULTS OF PREGNANEDIOL ESTIMATIONS OF ONE 48 HOUR SAMPLE OBTAINED FROM EACH OF THE SUBJECTS OF GROUP (b)



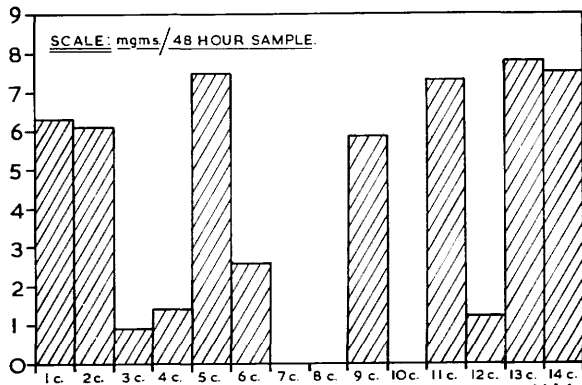


**FIG. XXIV.**

DIAGRAMATIC REPRESENTATION OF THE RESULTS OF PREGNANEDIOL ESTIMATIONS OF TWO 48 HOUR SAMPLES OF URINE OBTAINED FROM EACH OF THE SUBJECTS OF GROUP (c). The first sample was obtained during the first half of the menstrual cycle, the second during the second half of the menstrual cycle.



VALUES OBTAINED DURING THE FIRST HALF OF THE MENSTRUAL CYCLE.



VALUES OBTAINED DURING THE SECOND HALF OF THE MENSTRUAL CYCLE.

Table XXIV

in which the subjects of group (a) are classified as (1) Probably Ovulating, (2) Possibly Ovulating or (3) Probably Not Ovulating, on the basis of their temperature records considered in conjunction with the values obtained from Pregnanediol Estimations of two 48-hour samples of urine, these having been obtained respectively during the low and high temperature phases of the cycle (if diphasic), or merely at two distinct times separated by at least two weeks (if the temperature record lacked any discernible pattern).

Subjects whose Temperature Record was Diphasic or Probably Diphasic

<u>No.</u>	<u>Initials</u>	<u>Pattern of Temperature Record</u>	<u>Amount of Pregnanediol in 48-hr. sample of urine (low temperature phase of cycle)</u>	<u>Amount of Pregnanediol in 48-hr. sample of urine (high temperature phase of cycle)</u>	<u>Classification of subject as Probably Ovulating, Possibly Ovulating or Probably Not Ovulating</u>
1a	Mrs. B.D.	Diphasic	.26 mgms.	14.75 mgms.	Prob.Ovulating
2a	" M.M.	Do.	Nil	12.75 "	Do.
6a	" J.C.(K)	Prob. Diphasic	.8 "	2.08 "	Do.
7a	" M.McD	Diphasic	Nil	5.7 "	Do.
8a	" J.C.(B)	Do.	Nil	6.2 "	Do.
9a	" M.H.(C)	Do.	Nil	7.6 "	Do.
10a	" H.T.	Do.	Nil	.9 "	Poss.Ovulating
11a	" C.K.	Prob. Diphasic	Nil	.66 "	Do.
12a	" A.R.	Diphasic	Nil	1 "	Do.
13a	" M.H.(T)	Do.	.15 mgms.	3.78 "	Prob.Ovulating
15a	" I.R.	Do.	Nil	Nil	Results Inconclusive
16a	" M.McI	Prob. Diphasic	Nil	.6 "	Poss.Ovulating
17a	" M.W.	Diphasic	.15 "	4.2 "	Prob.ovulating
18a	" H.P.	Do.	.28	.65 "	Poss.Ovulating

Subjects whose Temperature Record was Diphasic or Probably Diphasic

<u>No.</u>	<u>Initials</u>	<u>Pattern of Temperature Record</u>	<u>Amount of Pregnane-diol in 48 hr. sample of urine (low temperature phase of cycle)</u>	<u>Amount of Pregnane-diol in 48 hr. sample of urine (high temperature phase of cycle)</u>	<u>Classification of subject as Probably Ovulating, Possibly Ovulating or Probably Not Ovulating</u>
19a	Mrs. E.C.	Diphasic	Nil	.9 mgms.	Poss. Ovulating
20a	" J. O'N	Prob. Diphasic	Nil	.84 "	Poss. Ovulating
21a	" M.D.	Do.	Nil	3.8 "	Prob. Ovulating

Subjects whose Temperature Record was Monophasic

3a	Mrs. J.F.(D)	Monophasic	Nil	Nil	Prob. Not Ovulating
4a	" E.D.	Do.	Nil	Nil	Do.
5a	" J.F.(B)	Do.	Nil	Nil	Do.
14a	" J.S.	Do.	Nil	Nil	Do.
22a	" J.B.	Do.	Nil	Nil	Do.
23a	Miss M.S.	Do.	Nil	Nil	Do.
24a	Mrs. M.C.	Do.	Nil	Nil	Do.

DISCUSSION

Before commenting on the results of this investigation, it is necessary to recognise the limitations of the method by which they were obtained.

Although its general convenience in the clinical measurement of urinary pregnanediol is widely recognised, the de Watteville method of 1948 cannot be said to possess either the sensitivity or the specificity of the method described by Klopper, Michie and Brown in 1955.

While it is unfortunate that the pregnanediol estimations of this investigation could not be undertaken by the Klopper method and the results thus acquire a greater quantitative precision, it is clear that the values obtained show a distinct trend the significance of which is not destroyed by what are now recognised as the inherent minor inaccuracies of the de Watteville method.

In a personal communication Klopper (1956), while indicating the reasons for the relative insensitivity of the de Watteville and other methods when compared with his own, particularly for the detection of small (e.g. extragonadal) amounts of pregnanediol in a 48-hour sample of urine, nevertheless acknowledges/

acknowledges its usefulness for the pregnanediol estimation of samples of urine obtained during pregnancy or the luteal phase of the cycle. Though emphasising that the actual mathematical values obtained in this investigation might, on the grounds of precision be criticised by the scientific purist, Klopper readily concedes the significance of the general tendencies shown in these results, and states that the de Watteville method may be accepted as "reasonably "accurate down to values of about the order of 2 mgms."

With these observations in mind it is interesting to observe that, of the 24 women whose uterus and ovaries had both been removed, 23 (i.e. 95.8%) were found to have no pregnanediol when a random 48-hour sample of urine was estimated by the de Watteville method, and that the amount of pregnanediol obtained from the urine of the remaining subject was only .25 mgms.

Forty-eight hour samples of urine obtained from the normal subjects of group (c) during the first half of the menstrual cycle were also found, in 13 cases out of 14, to have no pregnanediol, though the sample obtained from the fourteenth subject (No. 6c) (just before the "thermal shift") was estimated to have .8 mgms. of pregnanediol.

It/

It is also to be noted that 48-hour samples of urine obtained from the women of group (a) either at random (if the temperature record was monophasic), or during the low temperature phase (if it was diphasic), produced a similar range of results, for in only 5 instances out of a possible 24 were positive values obtained (Nos. 1a, 6a, 13a, 17a and 18a), and of these the highest was .8 mgms.

When however one considers the results obtained from the same group of women (i.e. those of group (a)) during the high temperature phase of the cycle, a totally different order of values is to be found, for in no fewer than 10 of the 24 samples the estimated content of pregnanediol exceeded 2 mgms. and in 2 cases (Nos. 1a and 2a) it exceeded 10 mgms.

These figures have a close affinity with those obtained from the normal subjects of group (c) during the second half of the menstrual cycle for, as may be seen from a study of Table XXI values exceeding 2 mgms. were obtained from the 48-hour samples of urine of 8 of the 14 women of this group.

It is to be observed, therefore, that on every occasion on which other evidence indicated the absence of a corpus luteum the corresponding value obtained from the pregnanediol estimation of a 48-hour sample of urine was less than 1 mgm. and/

and in the great majority of cases was zero. The phrase "other evidence" as used here means:- (1) a history of bilateral oophorectomy, (2) the absence of a "thermal shift" in the temperature record, or (3) the absence of secretory changes in the endometria of the subjects of group (c).

In those cases however where the temperature record suggested continued ovulation and a 48-hour sample of urine was obtained during what was thought to be the luteal phase of the cycle, values exceeding 1 mgm. were frequently to be found, and in many instances, as may be observed by a glance at Tables XXI and XXIV that figure was often exceeded by several hundred per cent.

It would seem justifiable therefore to regard instances in which a diphasic temperature record was supported by a pregnanediol value in excess of 2 mgms. per 48-hour sample of urine (obtained during the high temperature phase of the cycle) as indicative of corpus luteum formation, and therefore as evidence of the probable continuance of ovulation.

On this basis 9 of the 24 subjects of group (a) have been classified as "probably ovulating" at the time of these investigations.

The temperature records of subjects 3c and 4c however demonstrated/

demonstrated that a 48-hour sample of urine obtained during what, on the basis of an endometrial biopsy, is assumed to be the luteal phase of the cycle, does not always produce pregnanediol values as high as 2 mgms. The value for subject 3c was .9 mgms. and that for subject 4c, 1.4 mgms.

This means that those subjects of group (a) whose temperature records exhibited a diphasic pattern but from whom pregnanediol values under 2 mgms. were obtained during the high temperature phase of the cycle, may also be examples of women who, at the time of these investigations, still continued to ovulate.

They have therefore been classified as "possibly ovulating".

Among the subjects of group (a) is one (No. 15a) whose temperature record exhibits the characteristics of the ovulating subject, although no pregnanediol was obtained from a 48-hour sample of urine collected from this woman during what appeared to be the high temperature phase of the cycle.

This discrepancy may be due to the operation of some fortuitous factor producing what appeared to be a diphasic temperature record.

Alternatively, it may be that the de Watteville method of pregnanediol estimation, the sensitivity of which is admittedly/



admittedly limited, failed to detect a relatively small amount of pregnanediol in this particular sample.

Because of the apparent contradiction between the results of the two investigations, it has been decided that this subject should not be placed in any of the three categories used in this investigation, the issue being left undecided.

#### SUMMARY and CONCLUSIONS

In this investigation, the conclusions reached in the previous one were subjected to an additional test of their validity.

This was considered desirable since some of the temperature records of the main investigation group (a) were difficult to classify.

It was thought that by performing pregnanediol estimations on 48-hour samples of urine obtained during the high and low temperature phases of the cycle, further evidence might be obtained concerning the probability or otherwise of corpus luteum formation, since the formation of/  
of/

of a corpus luteum would be expected to result in an appreciable liberation of progesterone by the ovary, and this in turn should be reflected in the pregnanediol content of the subject's urine.

A brief account is given of the history of pregnanediol and of the evidence for assuming it to be a metabolite of progesterone.

Previous attempts by Jones and Te Linde (1941) and by Davis and Fugo (1948) to assess ovarian function in recently hysterectomised women by means of urinary pregnanediol estimations are mentioned.

The method employed in this investigation is that of De Watteville, Borth and Gsell (1948). It is described in detail and its limitations discussed. The reasons for its employment are stated, and it is suggested that the recognised minor inaccuracies of this standard clinical method are not of sufficient degree to nullify the broad general trends that emerge in the results of this investigation.

Forty-eight hour samples of urine were obtained from the subjects of all three investigation groups and were estimated/

estimated quantitatively for pregnanediol by the method of de Watteville, Borth and Gsell.

The subjects of group (b) (i.e. those who had undergone hysterectomy with bilateral oophorectomy) each submitted one 48-hour sample of urine for estimation.

The subjects of group (c) (i.e. the normal group), each submitted two 48-hour samples of urine, one collected during the first half of the menstrual cycle and one during the second half.

The main investigation group (i.e. those subjects who had undergone hysterectomy with conservation of one or both ovaries) each submitted two samples of urine for estimation.

If the subject's temperature record was diphasic one sample was obtained during the low temperature phase and one during the high temperature phase.

Subjects of group (a) whose temperature records were monophasic submitted two samples of urine separated by an interval of at least two weeks.

The results obtained showed strong agreement between the subjects' temperature records and the values obtained from urinary pregnanediol estimation.

The/

The interpretation of the results is discussed in detail and an attempt has been made, on the basis of the results of this investigation and the preceding one, to classify the subjects of group (a) as (i) probably ovulating, (ii) possibly ovulating, or (iii) probably not ovulating.

On the evidence of this investigation and the preceding one, it was considered that ovulation was probably still occurring in 9 of the 24 subjects of group (a), that in another 7 there was the possibility of its continued occurrence, but that in the remaining 7 subjects ovulation probably did not then take place. The results in one case were considered to be inconclusive.

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**FOURTH INVESTIGATION**

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I N T R O D U C T I O N

The two previous investigations were designed to ascertain whether or not those ovaries which had been conserved at hysterectomy retained, after varying intervals of time, the capacity to produce the hormone progesterone. The tests were, in consequence, virtually tests of the continuance of ovulation, since progesterone is the hormone of the corpus luteum, and corpus luteum formation is the normal sequel of ovulation.

There is, however, evidence for the belief that the concluding years of reproductive life are characterised by "irregular follicle ripening phases, prolonged transitional phases and very frequently poor luteal phases" (Bonine 1950). Inability to demonstrate the production of progesterone does not therefore argue a complete cessation of activity on the part of the ovaries which may still be capable of producing oestrogen.

It seemed desirable therefore, to conduct an investigation into the state of oestrogen production in those ovaries which had been conserved at hysterectomy, and it/

it was with this object that the present test was undertaken.

### Rationale of the Investigation

The vaginal epithelium has a basal layer of cells which are nourished by underlying capillaries. As this layer proliferates additional cells are formed and pushed upwards towards the surface. The cells change in character as they migrate from the basal layer. The more superficial the location the flatter become the cells, the clearer becomes the cytoplasm and the greater becomes the ratio of cytoplasm to nucleus. These changes are merely the physical manifestation of two operative factors which are (1) the pressure of adjacent cells and (2) the increase in distance from the source of nourishment.

Since the action of oestrogen on the upper portion of the vaginal epithelium is essentially that of a growth hormone (Novak 1941), promoting mitotic division of the basal cells and a resultant thickening of the epithelium, it follows that the greater the concentration of oestrogen, *ceteris paribus*, the greater will be the number of layers of/

of the vaginal epithelium, the farther will the superficial cells be separated from their supplies of oxygen and nourishment and the more degenerate will they become.

The main indications of degeneration of the superficial cells are that the cytoplasm becomes acidophylic and the nucleus pyknotic. These acidophylic cells with pyknotic nuclei are said to be "cornified" and the percentage of "cornified cells" among those desquamated from the epithelium of the upper part of the vagina may be regarded as a good index of the oestrogenic activity of the ovaries (Vincent Memorial Hospital, 1950).

This investigation is therefore an attempt to discover whether or not the desquamated vaginal cells of those women whose ovaries were conserved at hysterectomy exhibit cornification, and if so, how the cell picture differs from those of the two control groups.

For comparative purposes the percentage of cornified cells in a particular smear may be regarded not only as a qualitative, but as a quantitative, measure of the oestrogen in that particular subject (Papanicolaou 1936).



### The Layers of the Vaginal Epithelium

Authorities differ on the subject of the number of layers of cells to be distinguished in the vaginal epithelium (McLaren 1941). It is not therefore surprising that the terminology used by different workers to describe the various cellular components of the vaginal smear should also differ.

Most writers, including Papanicolaou (1933) distinguish three strata in the vaginal epithelium. These are (a) a deep stratum, (b) a middle stratum and (c) a superficial stratum.

The deep stratum which rests on the basement membrane consists of (1) a row of cylindrical cells standing side by side. They possess large, deeply staining nuclei and sparse basophilic cytoplasm. These constitute the germinal layer. (2) Immediately superficial to them are to be found two or three layers of rounded cells with relatively large nuclei which have chromatin nets that stain well with haematoxylin. Their cytoplasm is clear in comparison with the nucleus. (3) Several rows of similar morphology but larger in size are next encountered as one moves towards the surface. In severe atrophy of the vagina some of these/

these cells may come to occupy a superficial position, become detached and be found in vaginal smears.

The middle stratum represents a transitional stage between the deep and the superficial strata. Its cells are polygonal in shape and have a clear transparent cytoplasm with a nucleus which stains well while still retaining the chromatin network. In vaginal smears these cells are very conspicuous and form a large proportion of those designated as intermediate cells.

The superficial stratum is composed of layers of cells varying greatly in number according to the blood oestrogen level. All the cells of this stratum are flat and polygonal in shape. Many of their nuclei are pyknotic. Those cells which lie in proximity to the middle stratum are usually found to have a basophilic cytoplasm and a relatively small prepyknotic nucleus.

Nearer the surface one may encounter cells with a cytoplasm which is rich in eleidin and takes up an acidophylic stain. The nucleus is pyknotic. In the classification of vaginal smears these are given the name of cornified cells.

From/

From what has been written above, it will be obvious that the thickness of the vaginal epithelium and the character of its more superficial cells will vary not only at the different stages of a woman's life but also during the different phases of the menstrual cycle. Thus, at birth the vaginal epithelium shows considerable stratification due to the influence of the maternal oestrogen which passes through the placental barrier into the foetal circulation and exercises its influence on the vaginal epithelium (Frankel 1938). The effect, of course, is transitory and within a few weeks desquamation has removed the more superficial cells and the infantile vagina is distinguished by a low epithelium which shows no evidence of oestrogenic activity (Bonine 1950).

Immediately prior to puberty, with the production of oestrogen in the follicles of the ovary, greater stratification of the vaginal epithelium is to be found as well as some cornified superficial cells (Papanicolaou 1933).

Throughout reproductive life the vaginal epithelium faithfully reflects the various phases of the ovarian cycle.

As/

As the Graafian follicle ripens, its oestrogenic activity increases. This results in a thick vaginal epithelium with an abundance of superficial cells of the cornified type. Ovulation coincides with maximum cornification, after which relatively fewer cornified cells are observed.

As the menopause approaches, ovarian activity wanes, primary follicles are reduced in number and the

"functional levels regress gradually to an inactive state during which time the pubertal and pre-pubertal functional levels may be recognised"

(Bonine 1950).

With the cessation of menstruation the ovary may be said to be nearing the end of its career as an oestrogen-producing organ. Desquamation of the more superficial epithelial cells is no longer counteracted by a rich compensatory proliferation of basal cells. Intermediate cells come to be situated more superficially and, with the passing of time, the atrophy becomes more pronounced and even deep cells may come to occupy a superficial position and be desquamated. A striking proof of the hypothesis which underlies this investigation is the fact that the continued administration of oestrogen to a postmenopausal woman/

woman whose vaginal smear exhibits a high proportion of deep cells, will produce a progressive diminution in their number, and their replacement by intermediate, and even by cornified, cells (Papanicolaou 1936).

As desquamation continues from birth until death the character of the vaginal smear may therefore be considered as a reliable indicator of current hormonal activity.

#### PREVIOUS INVESTIGATIONS

(a)

De Allende and Orlas (1950C) in their "Cytology of the Human Vagina" devote part of a chapter to a description of the types of vaginal smear which they discovered in women who had previously undergone hysterectomy with, and without, conservation of ovaries.

A study was made of the vaginal smears of four women, whose ages ranged from 34 to 46 years, and who had undergone the operation of subtotal hysterectomy with bilateral oophorectomy at times varying from four months to twelve years previously. No cornified cells were discovered in the/

the smears of this group, and deep cells which were a constant finding, varied from 2 to 25 per cent. The intermediate cell predominated and leucocytes and mucus were also in abundance.

The smears of two women, aged 42 and 44, who had undergone the operation of subtotal hysterectomy with conservation of both ovaries three and five years previously were also studied. Cornification peaks of 38 to 41 per cent were attained in these cases and deep cells were only occasionally found. De Allende and Orias, while classifying these cases as exhibiting "subnormal ovarian function" emphasise the fact that both women were of "relatively advanced age" and that they might therefore have ceased to ovulate. They conclude that the absence of the uterus "has no manifest influence upon the state of the vaginal epithelium".

Four patients, whose ages ranged from 27 to 44 years and who had undergone the operation of subtotal hysterectomy with unilateral oophorectomy within the previous five years, were the next subjects of investigation. The smears of these women gave a picture of "subnormal ovarian function", cornified/

cornified cells being present, though in smaller numbers than in the previous group.

From these investigations it would appear that the operation of subtotal hysterectomy does not seem to have an adverse influence on ovarian activity.

Unfortunately no women who had undergone total hysterectomy were studied, so that the effect of the more radical operation was not estimated.

This series is also rather small to justify generalisation.

(b)

Only one other instance was discovered of vaginal smears having been used in an attempt to assess the ovarian function of women who had undergone the operation of hysterectomy.

The results of this work, which was performed by Bancroft-Livingston (1954), were published while the present investigation was being undertaken.

Bancroft-Livingston in his investigation of 353 cases of hysterectomy with conservation of ovaries (all of whom were/

were under the age of 45 at the time of operation) found that 95% of those subjects who were investigated within three years of the date of the removal of the uterus, showed "active" vaginal smears.

A control series consisting of 95 women of comparable age group, from whom neither uterus nor ovaries had been removed, yielded similar results.

He also investigated 215 women upon whom hysterectomy had been performed after the age of 45 years had been reached. The results obtained were compared with those of a control group of 97 "normal" women of comparable age, and once again no significant difference was detected between the results of the two groups, though the percentage of "active" vaginal smears in these groups was much lower than in the two just mentioned.

Bancroft-Livingston concluded that

"following hysterectomy at whatever age, preserved ovarian tissue continues to function for considerable periods of time".

In the published report of this work, it is significant (and unfortunate) that its author did not investigate, as a second control group, a number of women of the appropriate age group from whom both uterus and ovaries had been removed.



M E T H O D

Vaginal smears were taken from women of all three groups. Those subjects whose ovaries had been conserved when hysterectomy was performed had smears taken daily for four weeks. Those whose ovaries had been removed together with the uterus, had smears taken on alternate days over a similar period, and from the second control group (i.e. that consisting of normal women) vaginal smears were taken daily throughout one menstrual cycle.

A glass pipette was introduced as deeply as possible into the vagina, so that the desquamated material collected might be that shed by the upper third of the vaginal epithelium. This is important, as the upper part of the vagina is of different embryological development from the lower part, and responds in a more pronounced fashion to oestrogenic stimulation. The pipette was then slowly withdrawn while its tip was in contact with the vaginal wall for a distance of about three centimetres, the bulb being gradually released.

The material thus obtained was next deposited on a glass slide without the pipette actually coming into contact with the/

the slide.

As the smear must not be allowed to dry, the slide was immediately "fixed" in a solution consisting of equal parts of ether and 95% alcohol. Fixing was achieved by keeping the slide for a minimum of three minutes in the above solution.

Although de Allende and Orias (1950A) state that smears may "remain in the fixing fluid for a month and possibly "longer without harm", staining was usually undertaken within a few hours.

The staining method employed was that in use at the Department of Obstetrics and Gynaecology of the University of Edinburgh.

The details are as follows:-

The slide was initially dipped ten times in 95% alcohol, 70% alcohol, 50% alcohol and distilled water successively, after which it was immersed for five minutes in Harris Alum Haematoxylin. It was then placed in running water for one minute, and thereupon dipped five times in .5% hydrochloric acid. It was next replaced in running water for a further four minutes, after which it lay in a solution of  $\frac{1}{2}\%$  lithium carbonate/

carbonate for one minute. Thereafter it was returned to the running water for one minute and dipped successively ten times in each of the following:- 50% alcohol, 70% alcohol and 95% alcohol, prior to its immersion in the stain Orange G.6 for one minute.

After this it was dipped ten times, twice consecutively, in 95% alcohol before being placed in the stain known as E.A.50 for a minute and a half.

Finally, it was dipped ten times in three consecutive solutions of 95% alcohol, dehydrated with absolute alcohol, cleared in xylol and mounted in Canada balsam.

Each smear was then examined under the microscope, and the percentage of each of the three main types of epithelial cell was determined on the basis of a total count of 400 cells.

### The Cells of the Vaginal Smear

As the term "deep cell", "intermediate cell" and "cornified cell" have slightly different meanings when used by different people, each will now be defined to illustrate its usage in the present context and to avoid ambiguity.

#### The Cornified Cell

A cornified cell is understood to represent the

"maximum differentiation of the cells of the vaginal epithelium under the action of the oestrogenic hormones"

(De Allende 1950B).

It is a large, thin polygonal cell with a clear homogeneous cytoplasm which is usually, but not necessarily, acidophylic. Since it is known that a local infection can produce a smear rich in acidophylic cells (Kernodle 1948), and since staining technique varies considerably, it was decided to follow the principle laid down by the cytologists of the Vincent Memorial Hospital (1950) who designate cells as "cornified" on the basis of nuclear changes alone. The criterion of cornification is thus pyknosis of the nucleus/

nucleus, and any superficial cell with a pyknotic nucleus has been classified as a cornified cell irrespective of the colour of its cytoplasm.

### The Intermediate Cell

The term intermediate cell, as used in this investigation, is applied to any cell of the vaginal epithelium which is obviously not a deep cell and yet does not conform to the above definition of a cornified cell.

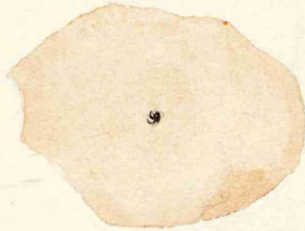
These cells therefore vary in size and morphology, some being almost oval and possessing a relatively large vesicular nucleus, while others are thin and polygonal and bear a close resemblance to the cornified type of cell except that the nucleus is not pyknotic. While the cytoplasm of most intermediate cells will be found to be basophilic, an acidophylic cytoplasm is a not infrequent finding, particularly where there is a vaginal infection. The nucleus may sometimes be found to stain deeply but its chromatin has not yet condensed to the solid black mass which denotes pyknosis.

### The Deep Cell

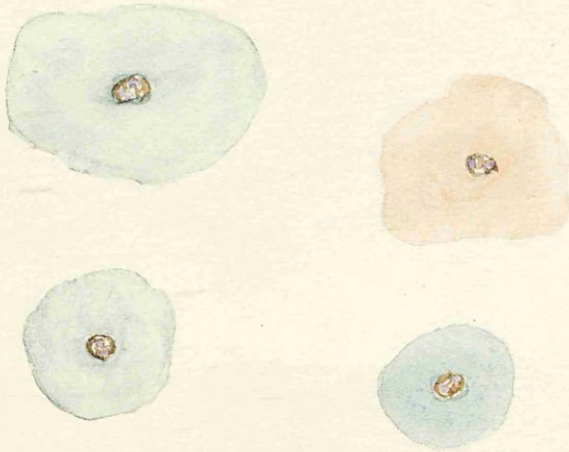
The deep cell is smaller than the intermediate cell, though larger than a polymorphonuclear leucocyte. It is somewhat rounded (or oval) in shape, the cell boundaries being precisely defined. The large centrally situated nucleus, regular in outline and containing a well differentiated network of chromatin, stains deeply with basic dyes. The ratio of the diameter of the cell to the diameter of the nucleus is about two to one. Where the cytoplasm has increased in size relative to the nucleus and the ratio has increased above that just mentioned, the cell has been classified as of the intermediate variety.

Deep cells are infrequently found in the premenopausal vaginal smear of the normally ovulating and menstruating woman, but become progressively more numerous as the oestrogenic powers of the ovary regress, and are a common component of the postmenopausal smear.

TYPICAL CORNIFIED CELL



INTERMEDIATE CELLS



DEEP CELL



Fig. XXV. illustrating the main types of cell to be found in vaginal smears.

Fig. XXVI. Microphotograph of vaginal smear obtained from Subject No. 9a (Mrs. M.M.(C)) on 15.9.54. The picture shows many cornified and intermediate cells. This subject had undergone hysterectomy with conservation of both ovaries more than two years previously.  
(Low Power View)

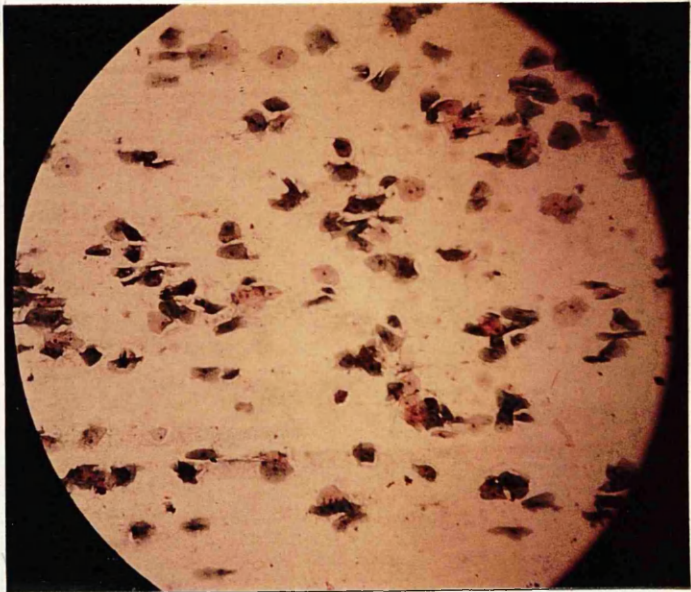




Fig. XXVII. Microphotograph of vaginal smear obtained from Subject No. 11b (Mrs. M.S.(K)) on 9.5.54. The picture shows no cornified cells. Only intermediate and deep cells were found in all smears obtained from this subject. (Low Power View)

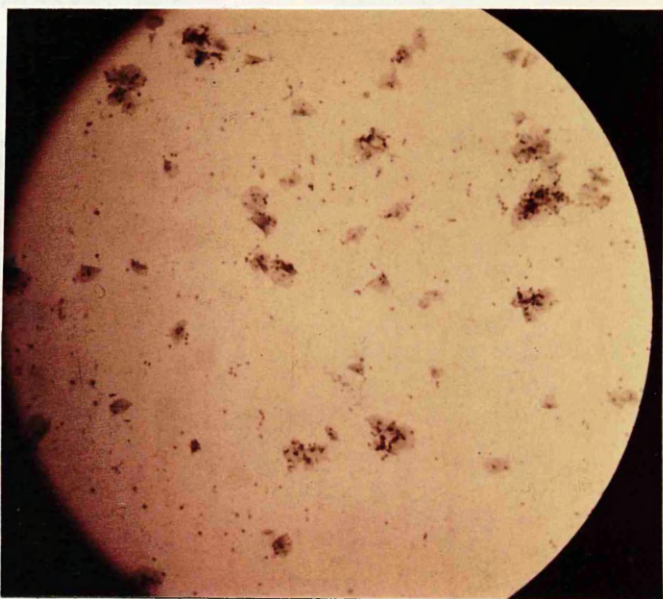


Fig. XXVIII. Microphotograph of vaginal smear obtained from Subject 22a (Mrs. J.B.) on 11.7.54. Although this woman had begun to complain of hot flushes it is to be noted that the smear shows a considerable number of cornified cells. This patient had undergone hysterectomy with unilateral oophorectomy more than four years previously. (Low power view)

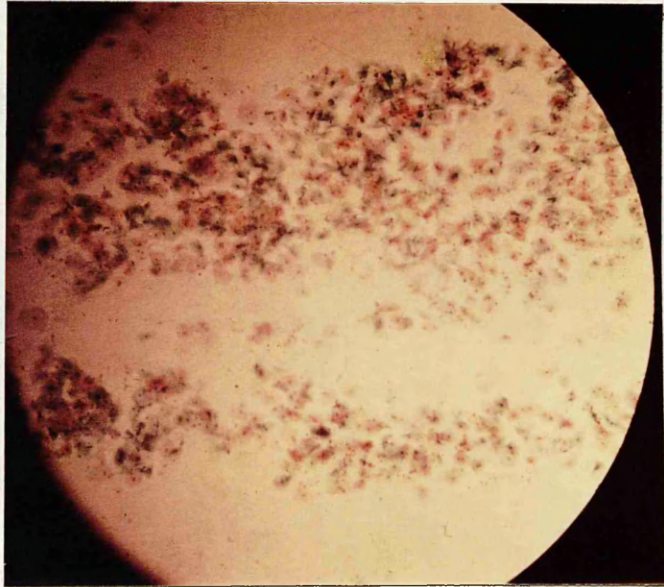




Fig. XXIX. Microphotograph of vaginal smear obtained from Subject 10a (Mrs. H.T.) on 15.5.54. The picture shows cornified and intermediate cells. The subject had undergone total hysterectomy with conservation of both ovaries more than a year previously.  
(Low power view)

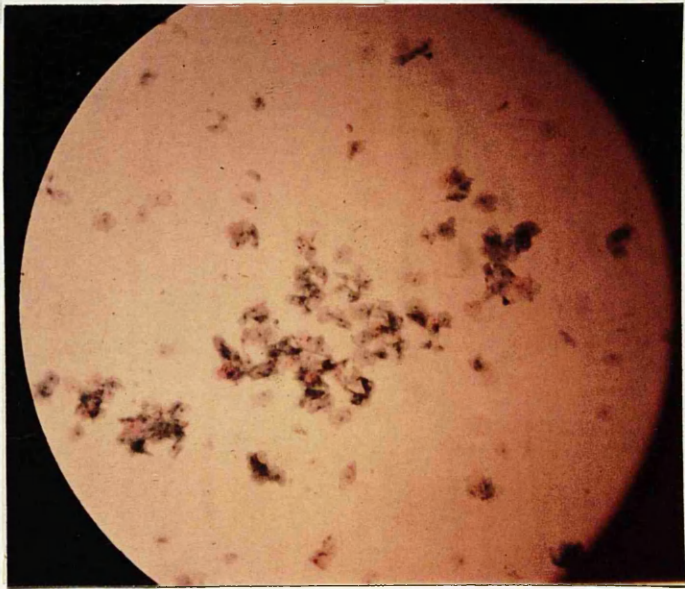


Fig. XXX. Microphotograph of smear obtained from Subject 9c (Mrs. G.S.) on 24.5.55. The picture shows cornified and intermediate cells. The subject had undergone no operation and was aged 39 6/12th years. X.160.

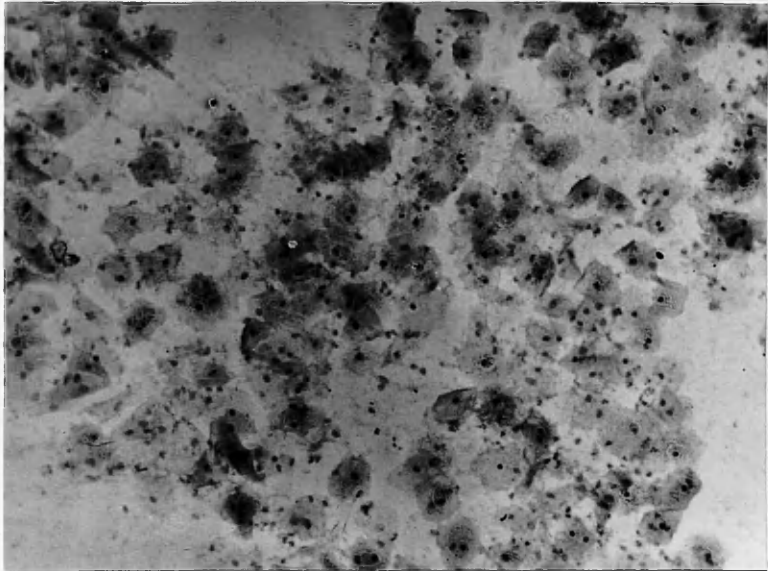
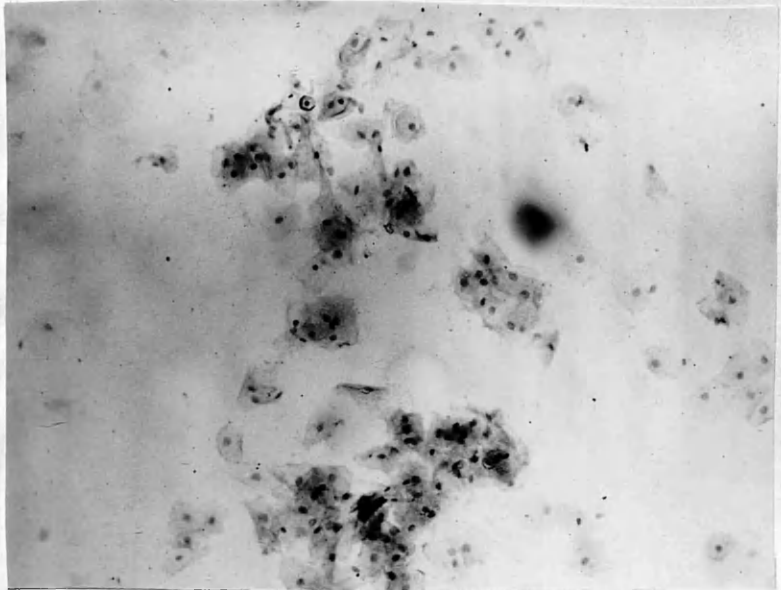


Fig. XXXI. Microphotograph of smear obtained from Subject 19a (Mrs. E.C.) on 10.5.54. The picture shows a few cornified and many intermediate cells. The subject had undergone subtotal hysterectomy with right oophorectomy more than two years previously. X.160.



RESULTS

The cornification curves of the 24 subjects of group (a) are here reproduced. In each case vaginal smears have been taken for about four consecutive weeks. The abscissae represent the days, the ordinates the percentages of cornified cells in the various smears.

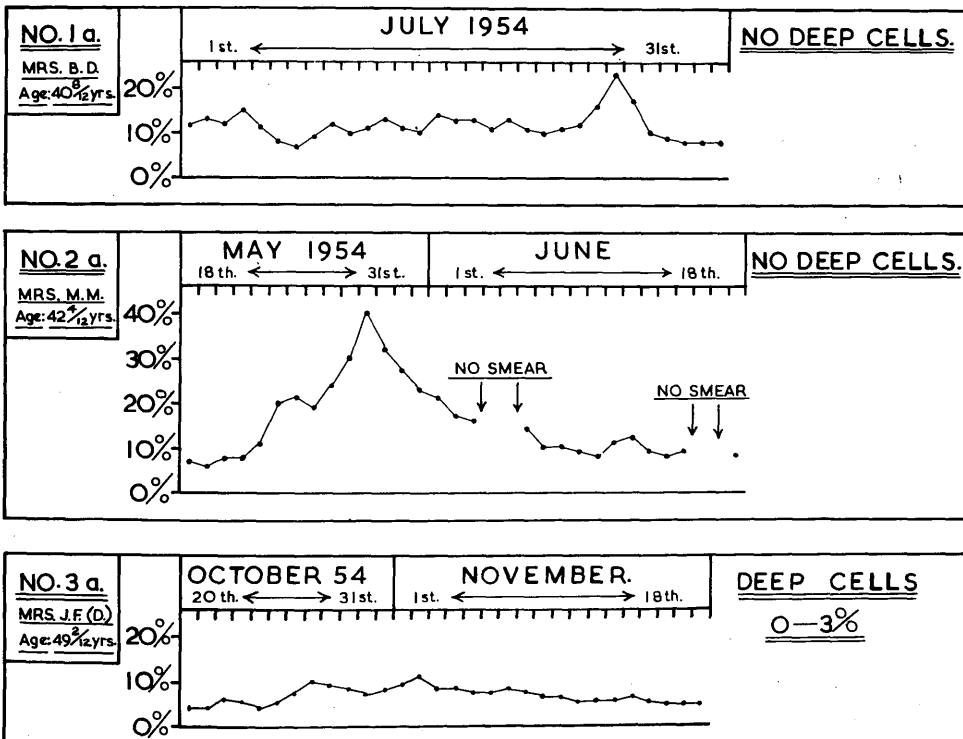


Fig. XXXII shows the cornification curves of subjects 1a, 2a and 3a.

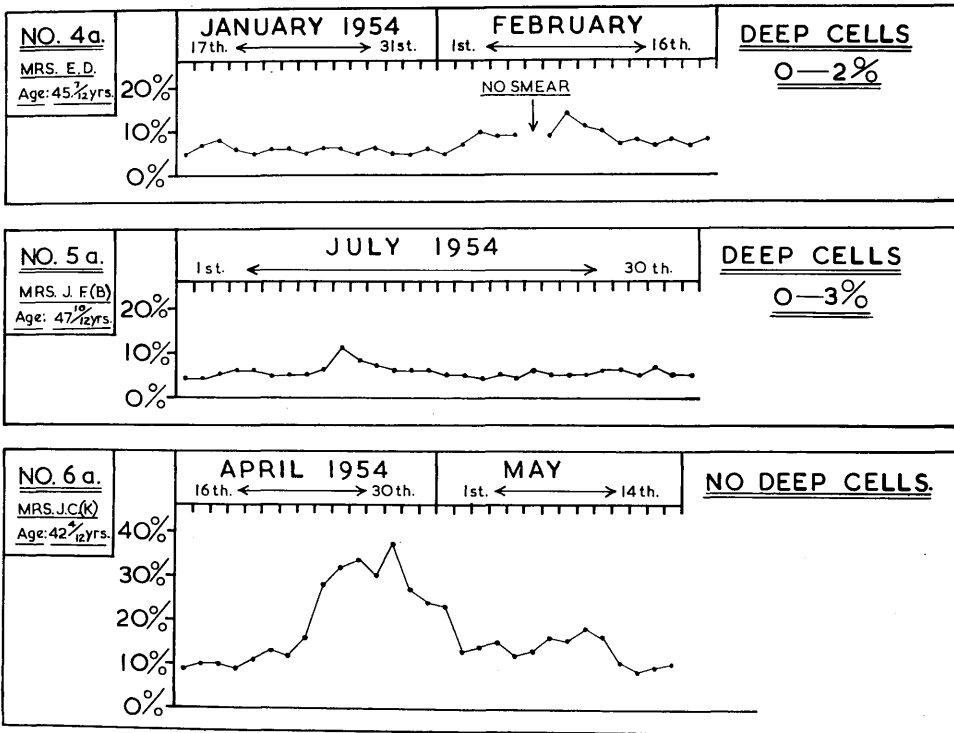


Fig. XXXIII shows the cornification curves of subjects 4a, 5a and 6a.

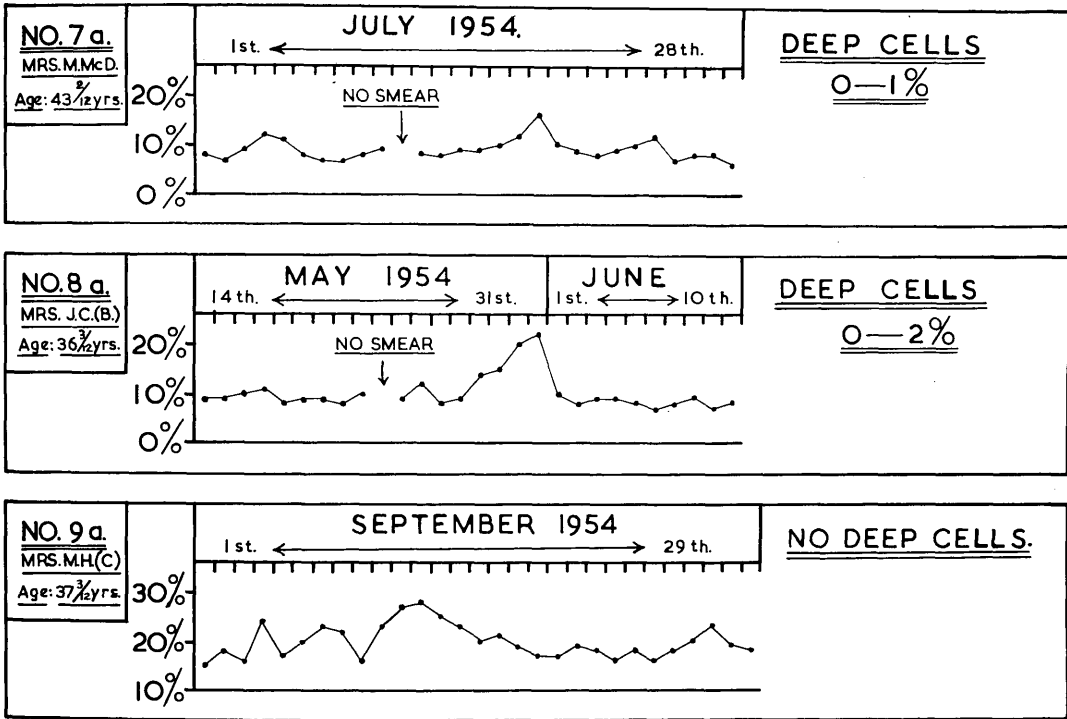


Fig. XXXIV shows the cornification curves of subjects 7a, 8a and 9a.



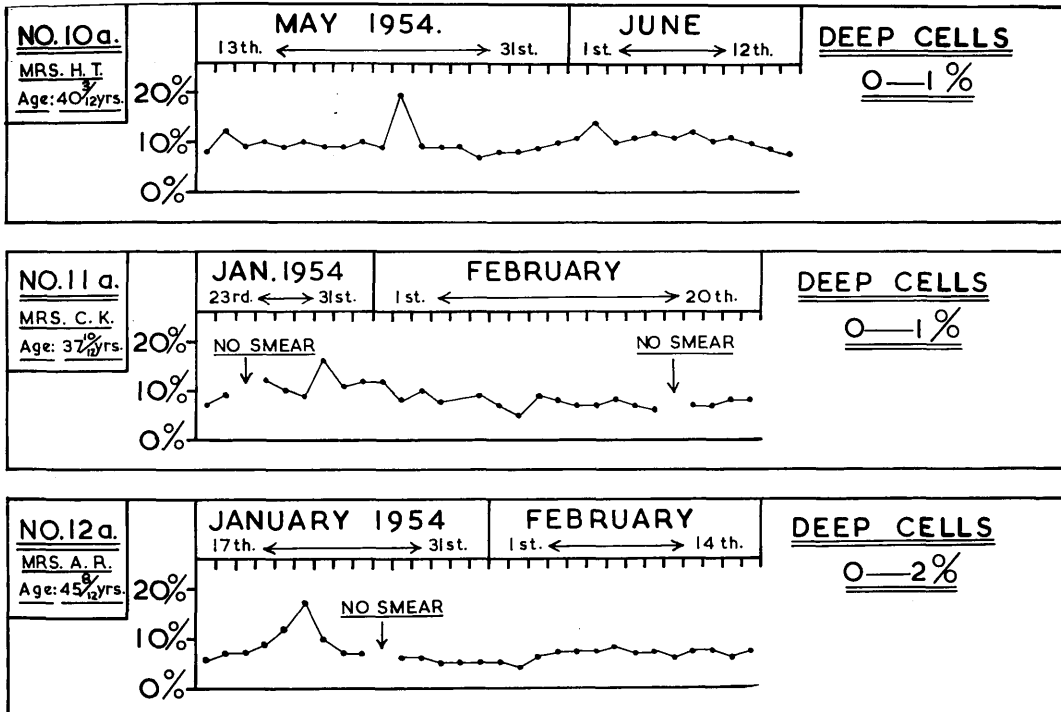


Fig. XXXV shows the cornification curves of subjects 10a, 11a and 12a.

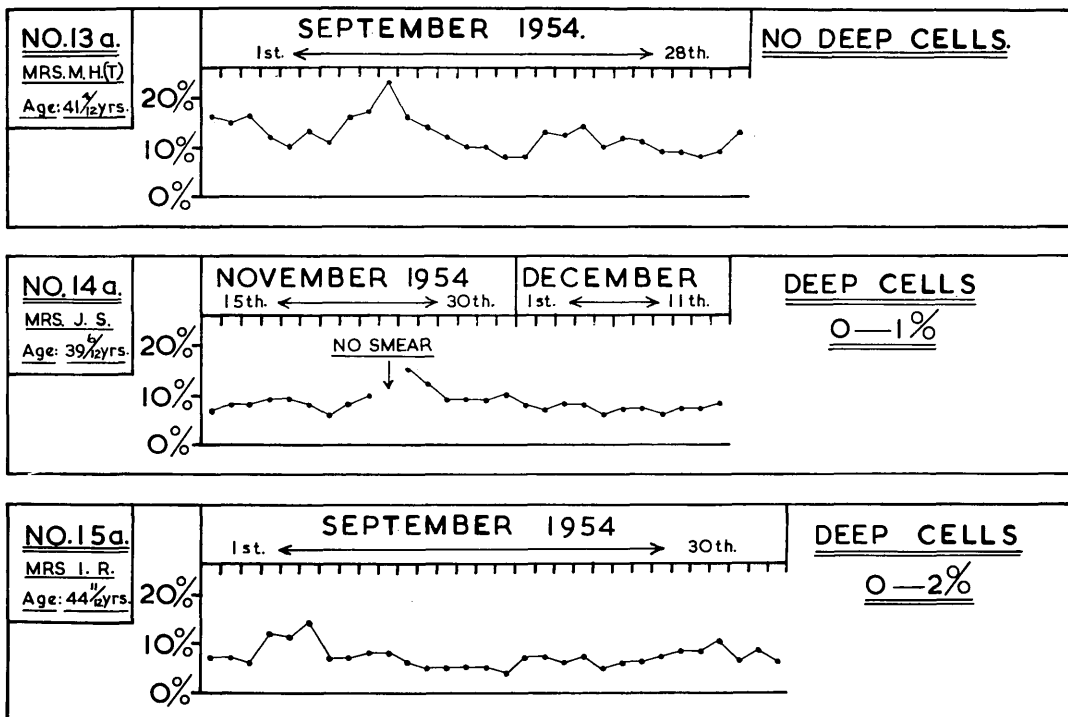


Fig. XXXVI shows the cornification curves of subjects 13a, 14a and 15a.

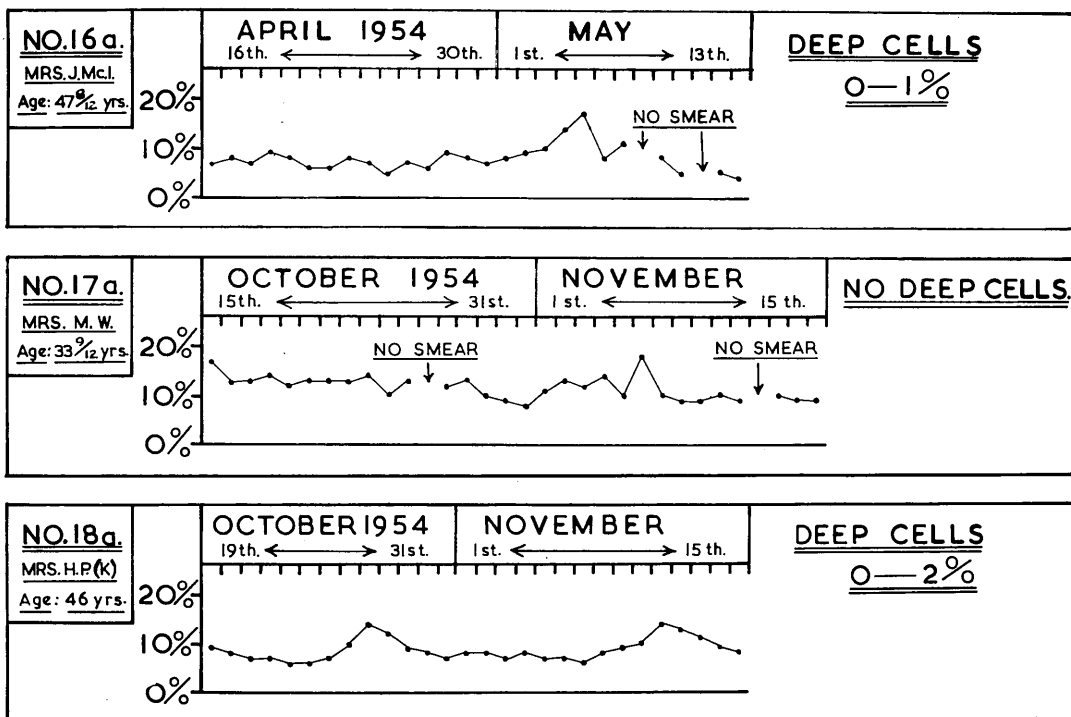


Fig. XXXVII shows the cornification curves of subjects 16a, 17a and 18a.

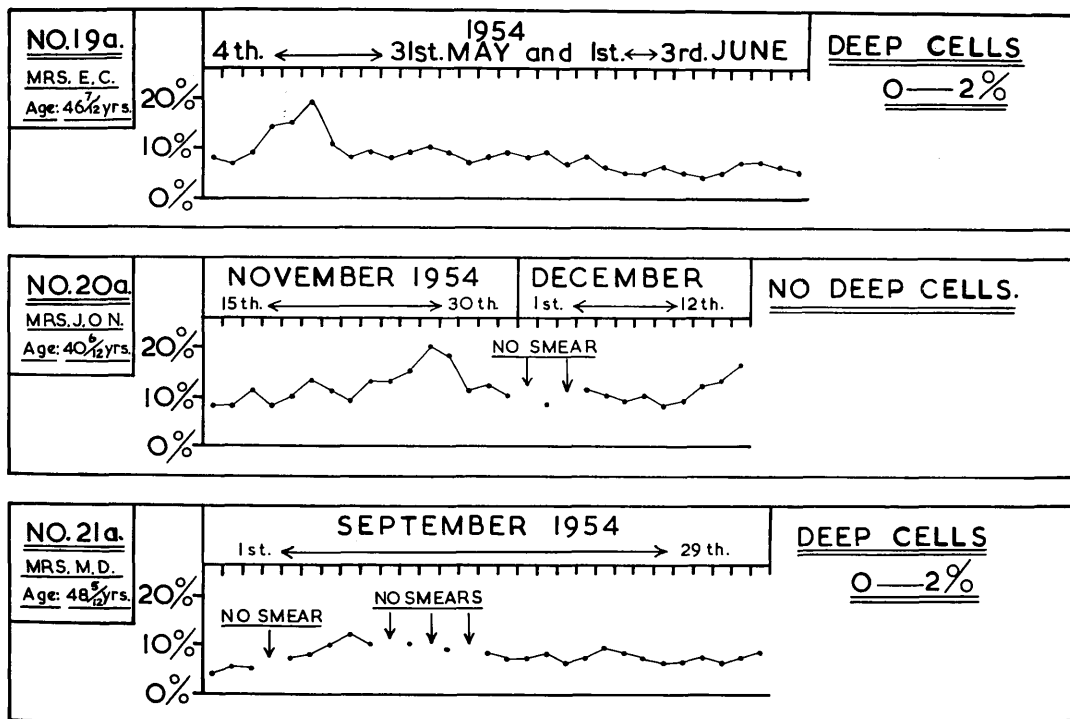


Fig. XXXVIII shows the cornification curves of subjects 19a, 20a and 21a.

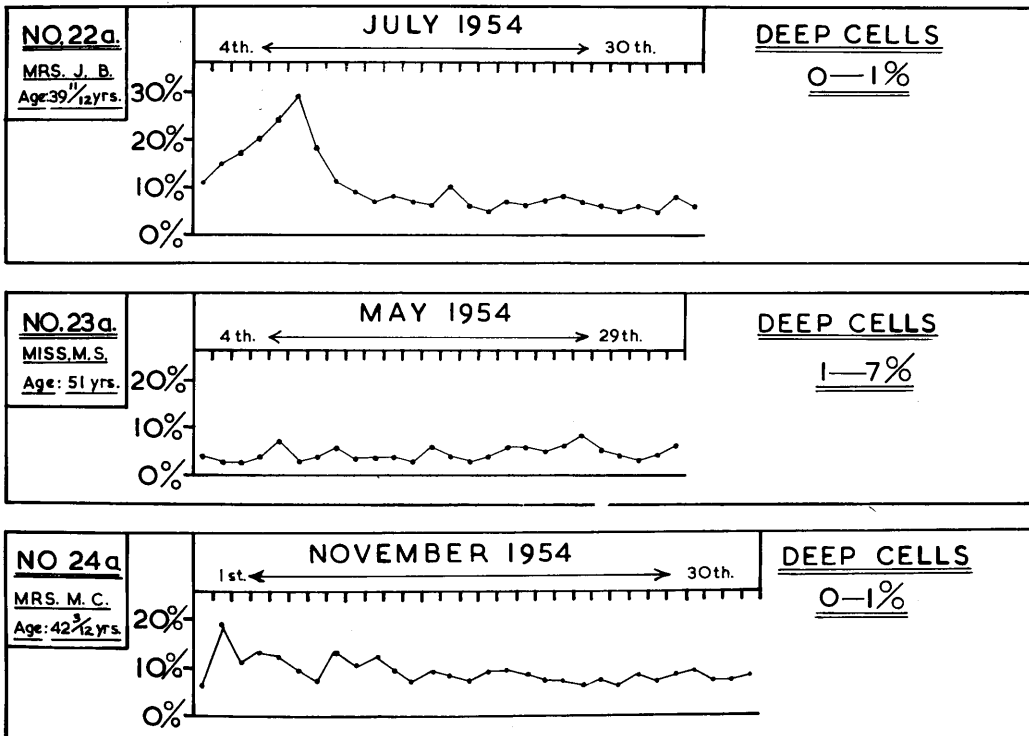


Fig. XXXIX shows the cornification curves of subjects 22a, 23a and 24a.

The curves of the deep cells of the 24 subjects of group (b) are here reproduced. Once again the vaginal smears have been taken for about four consecutive weeks. The abscissae represent the days and the ordinates the percentages of deep cells in the various smears.

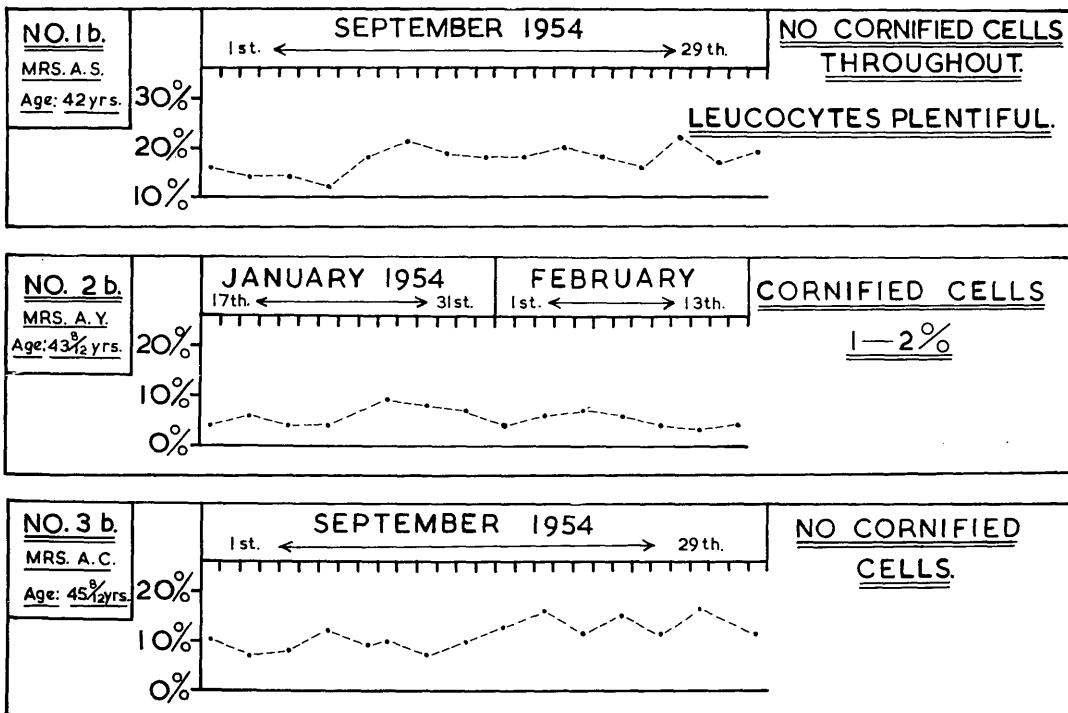


Fig. XL shows the deep cell curves of subjects 1b, 2b and 3b.

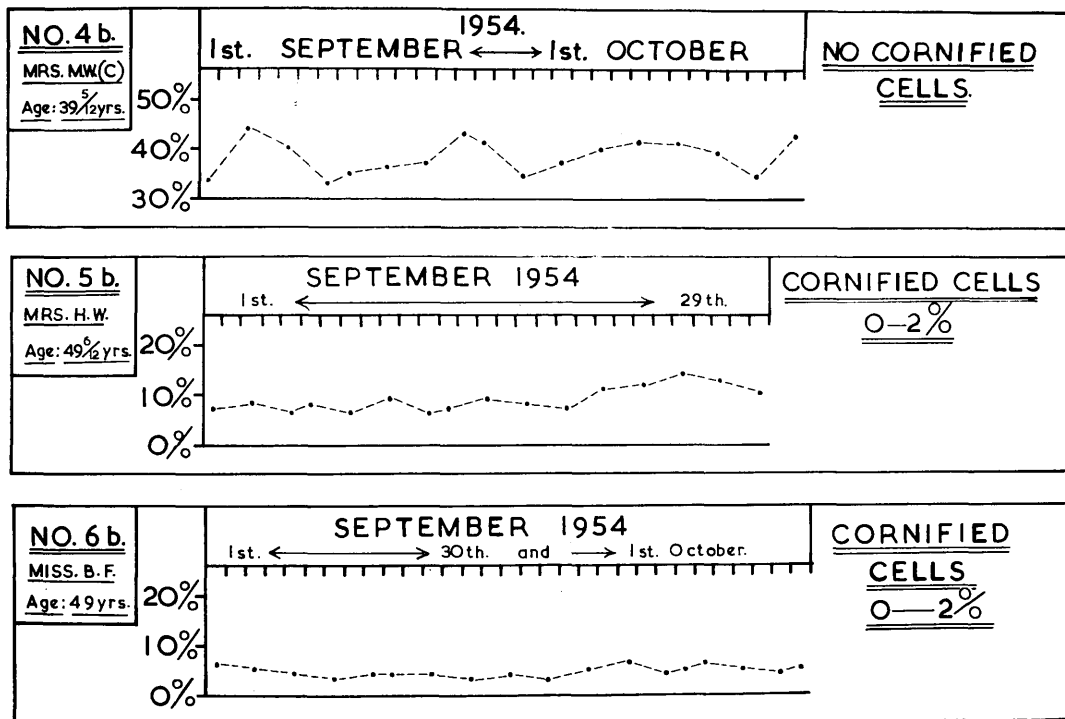


Fig. XLI shows the deep cell curves of subjects 4b, 5b and 6b.

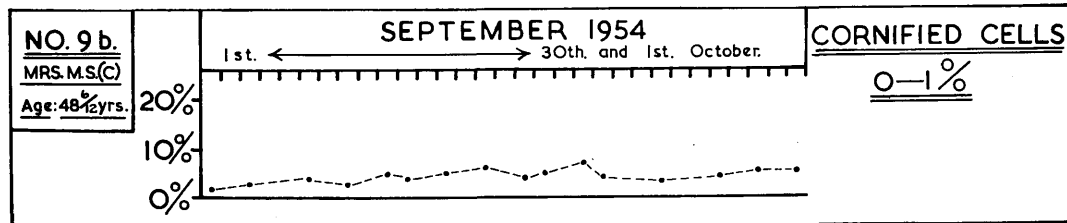
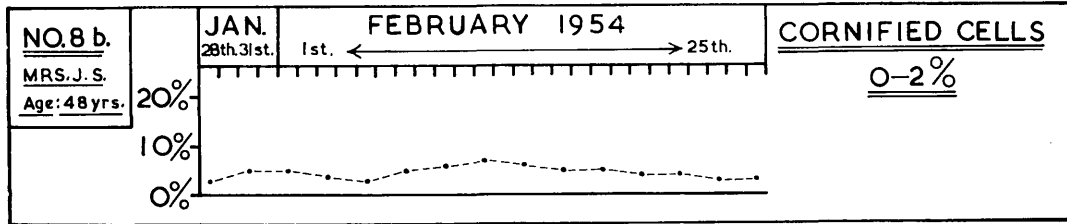
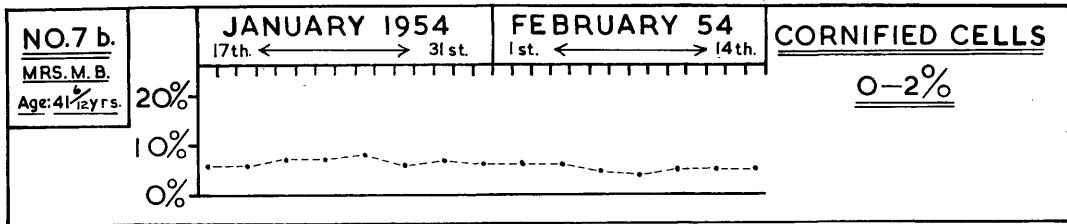


Fig. XLII shows the deep cell curves of subjects 7b, 8b, and 9b.



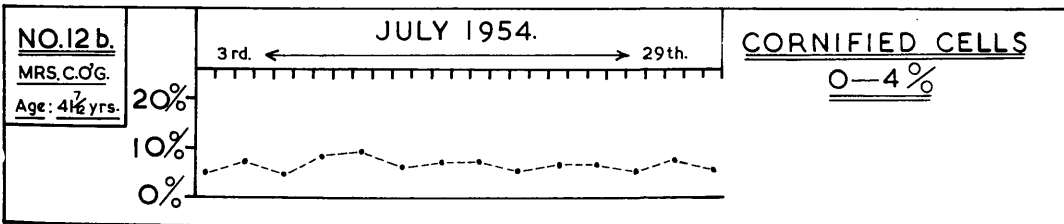
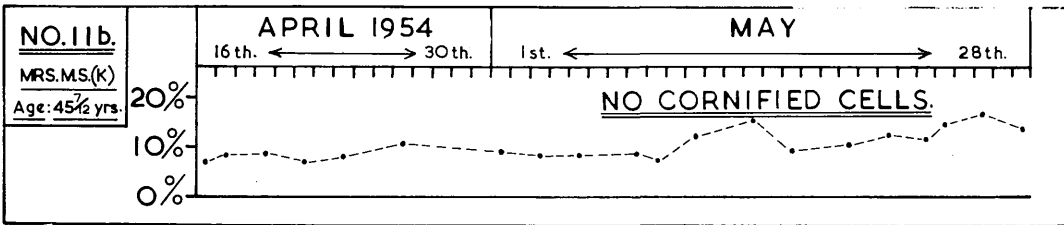
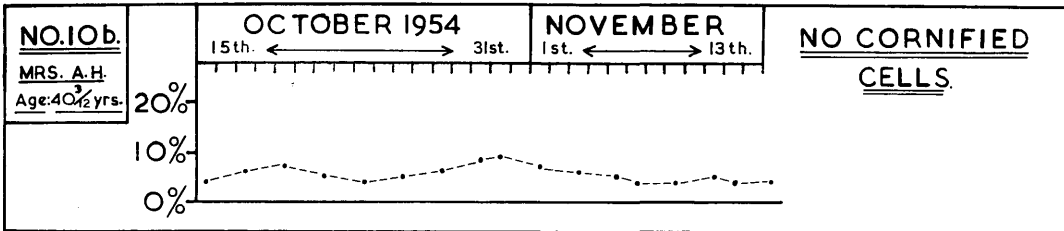


Fig. XLIII shows the deep cell curves of subjects 10b, 11b and 12b.

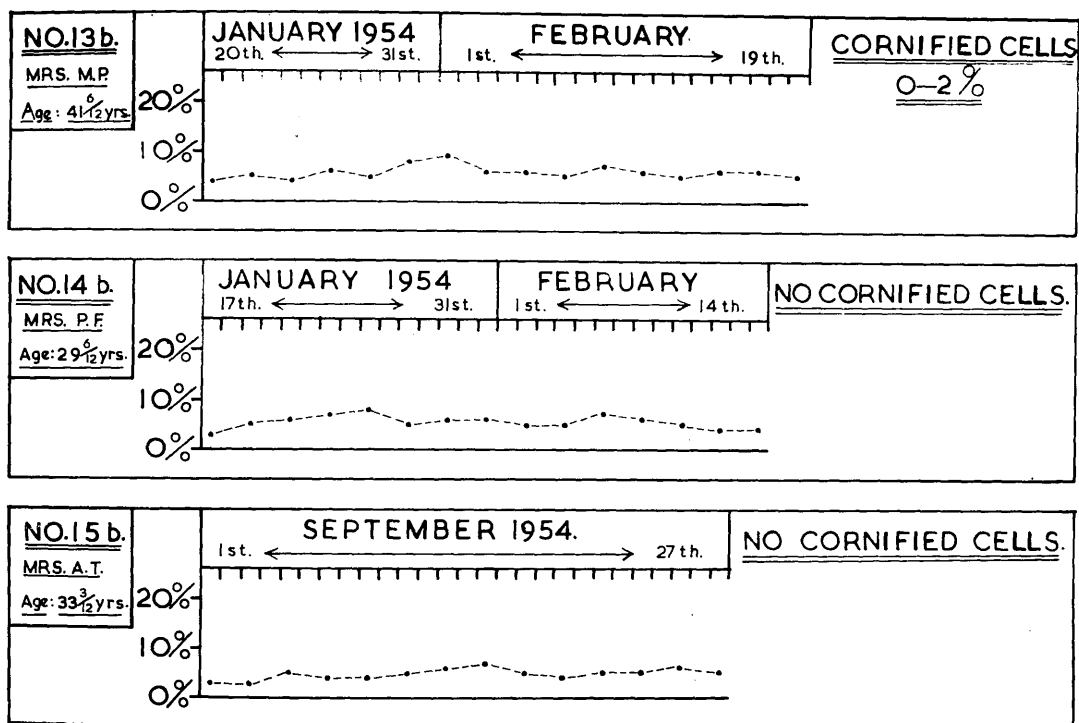


Fig. XLIV shows the deep cell curves of subjects 13b, 14b and 15b

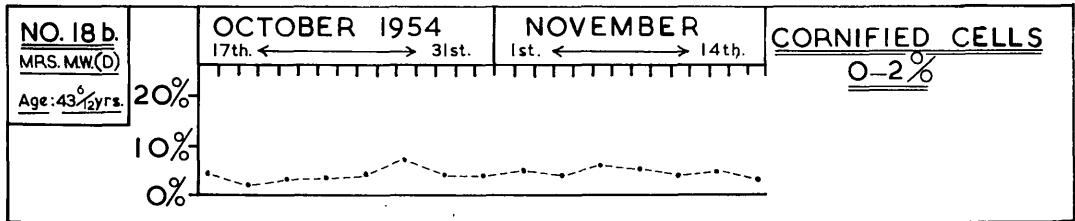
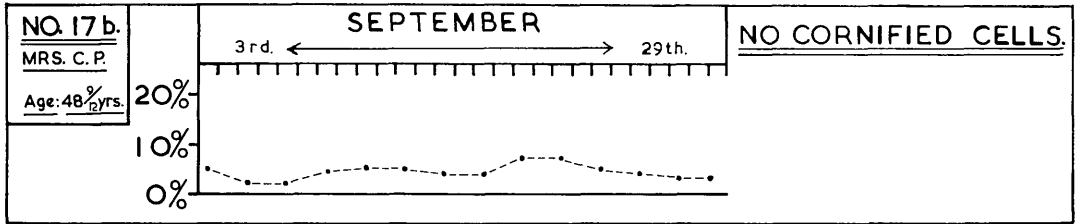
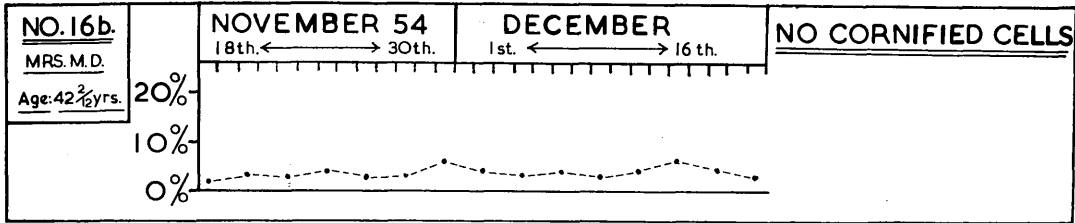


Fig. XLV shows the deep cell curves of subjects 16b, 17b and 18b.

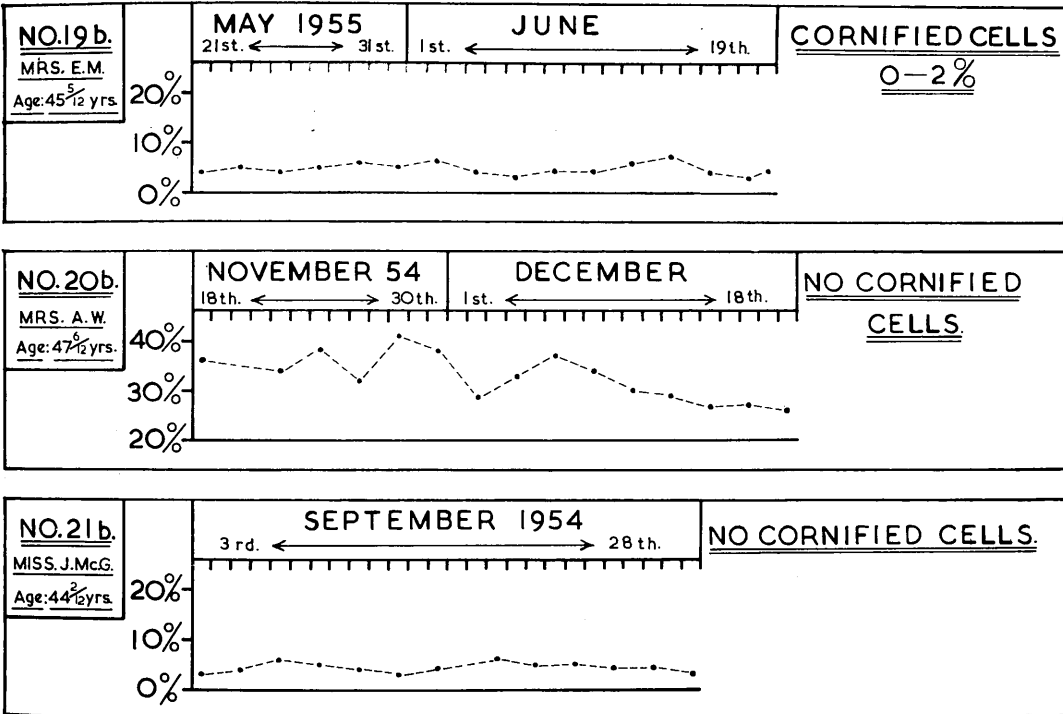


Fig. XLVI shows the deep cell curves of subjects 19b, 20b and 21b.

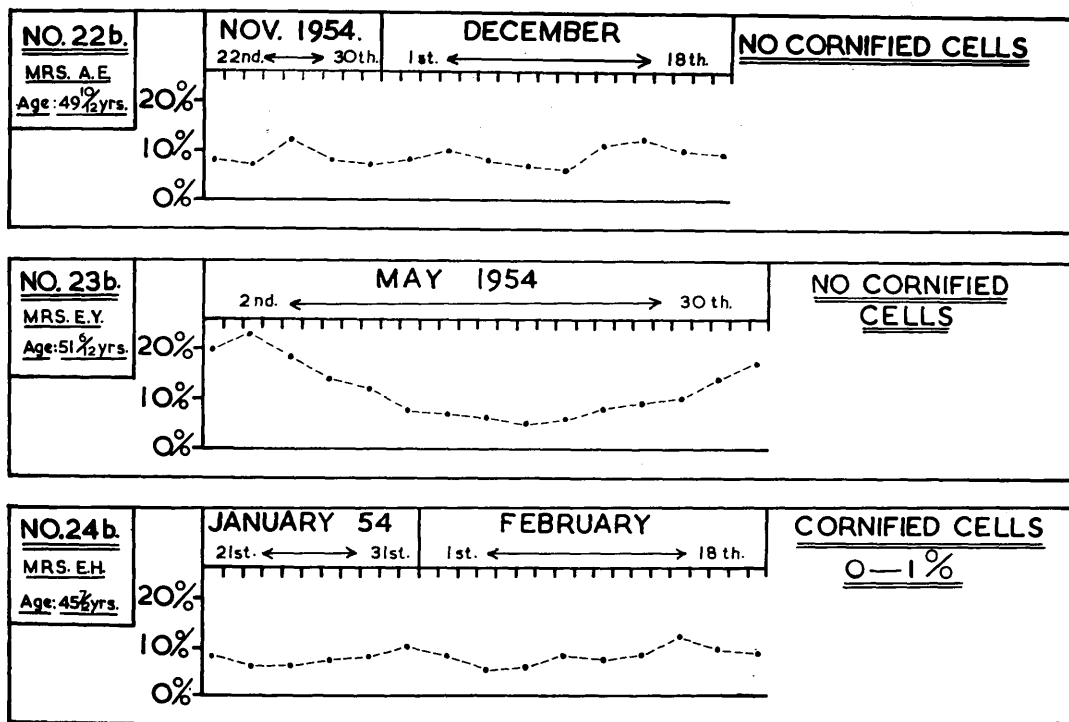


Fig. XLVII shows the deep cell curves of subjects 22b, 23b and 24b.

The cornification curves of the 14 subjects of group (c) are here reproduced. In each case, vaginal smears have been taken throughout one menstrual cycle, excluding the days on which there was vaginal bleeding. The abscissae represent the days, and the ordinates the percentages of cornified cells in the various smears.

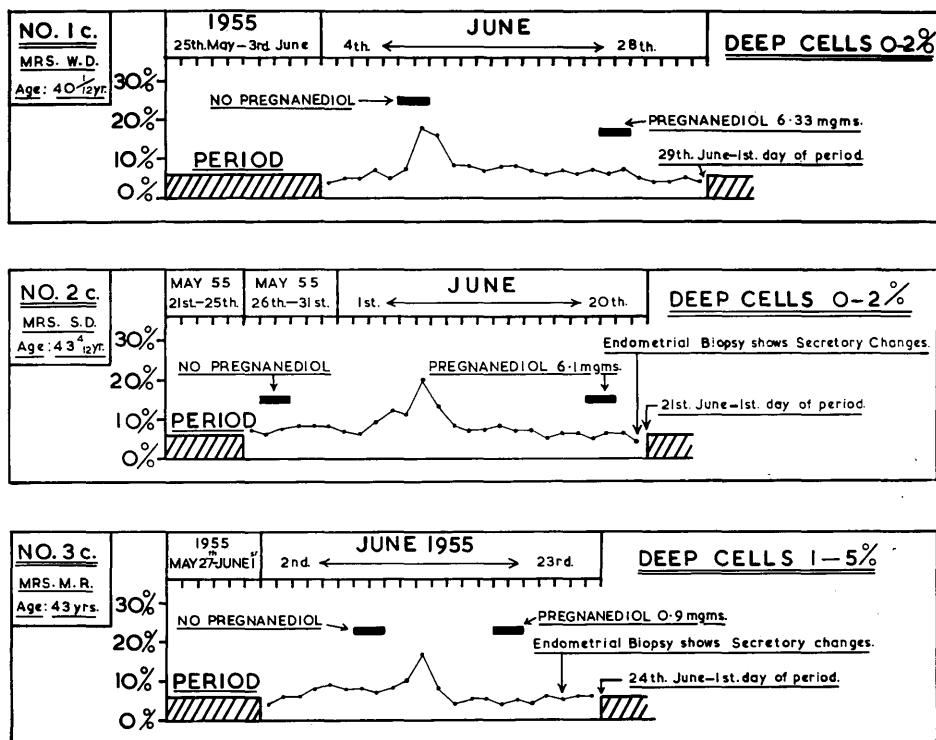


Fig. XLVIII shows the cornification curves of subjects 1c, 2c and 3c.

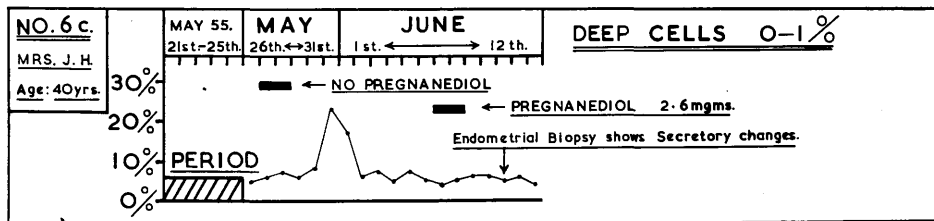
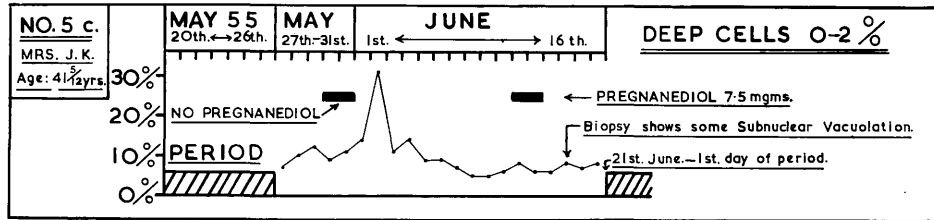
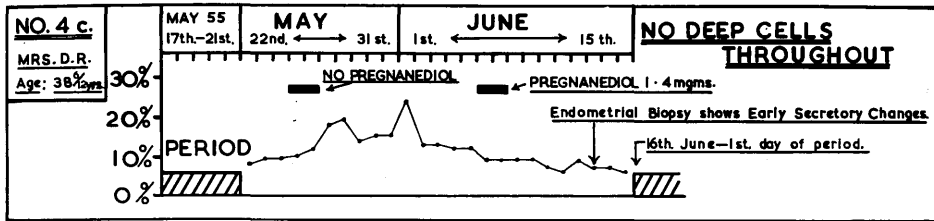


Fig. XLIX shows the cornification curves of subjects 4c, 5c and 6c.

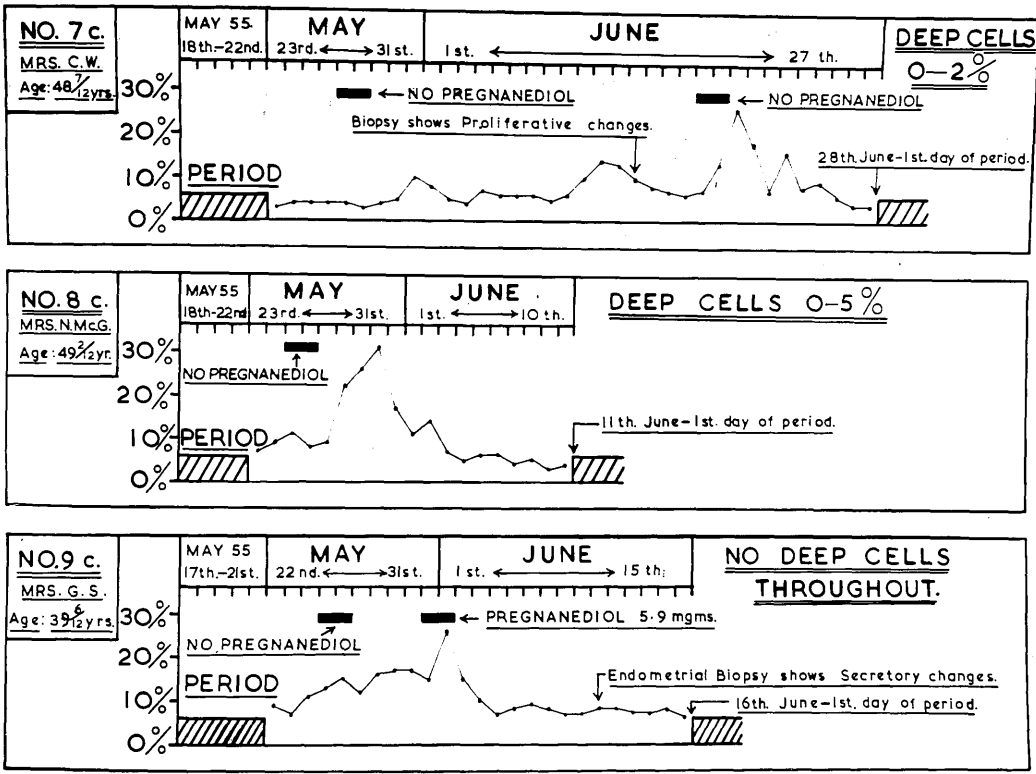


Fig. L shows the cornification curves of subjects 7c, 8c and 9c.



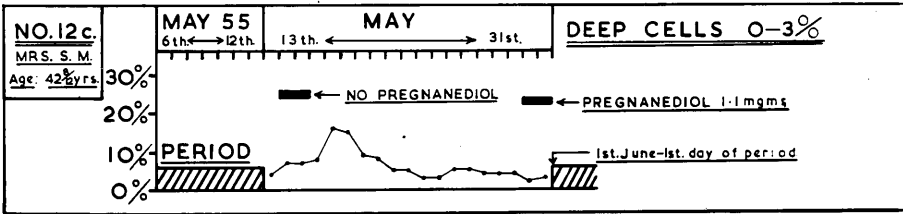
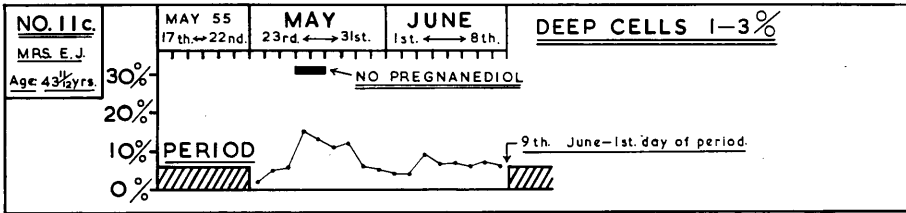
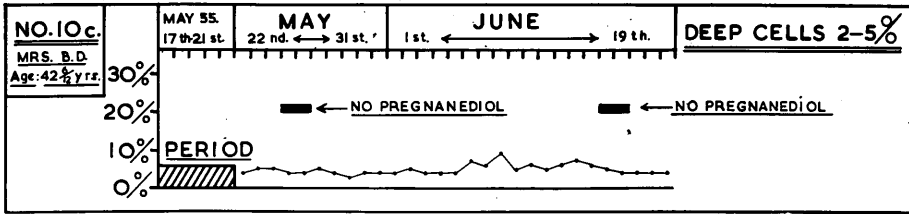


Fig. LI shows the cornification curves of subjects 10c, 11c and 12c.

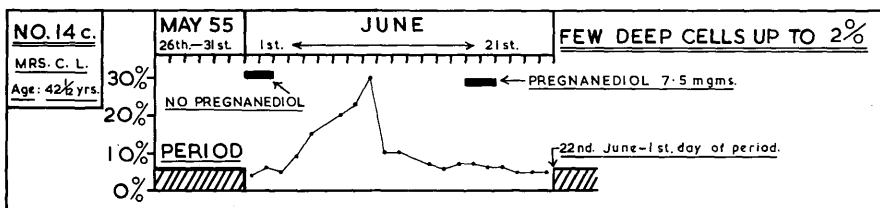
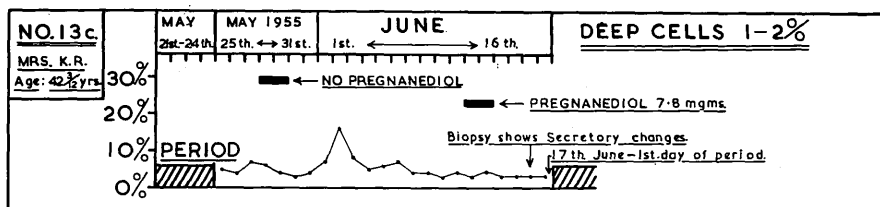


Fig. LII shows the cornification curves of subjects 13c and 14c.

Table XXV

Showing the main values of the Cytological Curves  
of the subjects of group (a)

<u>No.</u>	<u>Initials</u>	<u>Age at time of Investi- gation Years</u>	<u>Range of % of Cornified Cells dur- ing period of Investi- gation</u>	<u>Mean % of Cornified Cells dur- ing period of Investi- gation</u>	<u>Range of Deep Cells</u>
1a	Mrs. B.D.	40 8/12ths	7-23%	11.7%	Nil
2a	" M.M.	42 4/12ths	6-40%	15.3%	Nil
3a	" J.F.(D)	49 2/12ths	4-11%	6.5%	0-3%
4a	" E.D.	45 10/12ths	5-14%	7.2%	0-2%
5a	" J.B.(B)	46 10/12ths	4-11%	5.6%	0-3%
6a	" J.C.(K)	42 4/12ths	8-37%	17%	Nil
7a	" M.McD	43 2/12ths	7-16%	9.1%	0-1%
8a	" J.C.(B)	37 3/12ths	8-22%	10.2%	0-2%
9a	" M.H.(C)	37 3/12ths	15-28%	19.1%	Nil
10a	" H.T.	40 3/12ths	8-19%	9.5%	0-1%
11a	" C.K.	37 10/12ths	5-16%	8.7%	0-1%
12a	" A.R.	45 8/12ths	4-17%	7.1%	0-2%
13a	" M.H.(T)	41 4/12ths	8-23%	12.4%	Nil
14a	" J.S.	39 6/12ths	6-15%	7.7%	0-1%
15a	" I.R.	44 11/12ths	4-14%	7.1%	0-2%
16a	" J.McI	47 8/12ths	5-17%	8%	0-1%
17a	" M.W.	33 9/12ths	8-17%	11.7%	Nil
18a	" H.P.(K)	46	8-20%	8.7%	0-2%
19a	" E.C.	46 7/12ths	4-19%	8.1%	0-2%
20a	" J.O'N	40 6/12ths	8-20%	11.4%	Nil
21a	" M.D.	48 5/12ths	4-12%	7.5%	0-2%
22a	" J.B.	39 11/12ths	5-29%	11.5%	0-1%
23a	Miss M.S.	51	3-8%	4.6%	1-7%
24a	Mrs. M.C.	42 3/12ths	7-19%	8.9%	0-1%

Table XXVI

Showing the main values of the Cytological Curves of the subjects of group (b).

<u>No.</u>	<u>Initials</u>	<u>Age at time of Investi- gation Years</u>	<u>Range of % of Deep Cells dur- ing period of Investi- gation</u>	<u>Mean % of Deep Cells during period of Investi- gation</u>	<u>Range of Corni- fied Cells</u>	<u>Mean % of Cor- nified Cells</u>
1b	Mrs. A.S.	42	12-22%	17.5%	0%	0%
2b	" A.Y.	43 8/12ths	3-9%	5.4%	1-2%	1.3%
3b	" A.C.	45 8/12ths	7-16%	11.6%	0%	0%
4b	" M.W.(C)	39 5/12ths	33-44%	38.4%	0%	0%
5b	" H.W.	49 6/12ths	6-14%	8.8%	0-2%	1.1%
6b	Miss B.F.	49	3-6%	4.1%	0-2%	1.3%
7b	Mrs. M.B.	41 6/12ths	4-8%	6.7%	0-2%	1.2%
8b	" J.S.	48	3-7%	4.5%	0-2%	1.4%
9b	" M.S.(C)	50 6/12ths	2-7%	4.2%	0-1%	.4%
10b	" A.H.	40 3/12ths	4-9%	5.5%	0%	0%
11b	" M.S.	45 7/12ths	7-16%	10%	0%	0%
12b	" C.O'G	41 7/12ths	5-9%	6.3%	0-4%	1.8%
13b	" M.P.	41 6/12ths	4-9%	5.8%	0-2%	.9%
14b	" P.F.	29 6/12ths	3-8%	5.8%	0%	0%
15b	" A.T.	33 3/12ths	3-7%	4.8%	0%	0%
16b	" M.D.	42 2/12ths	2-6%	3.6%	0%	0%
17b	" C.P.	48 9/12ths	2-7%	4.3%	0%	0%
18b	" M.W.(D)	43 6/12ths	3-7%	4.1%	0-2%	.8%
19b	" E.M.	45 5/12ths	3-7%	5%	0-2%	1.1%
20b	" A.W.	47 6/12ths	26-41%	32.7%	0%	0%
21b	Miss J.McG	44 2/12ths	3-6%	4.3%	0%	0%
22b	Mrs. A.E.	49 10/12ths	7-12%	8.9%	0%	0%
23b	" E.Y.	51 6/12ths	5-23%	12.2%	0%	0%
24b	" E.H.	45 7/12ths	5-10%	7.7%	0-1%	.4%

Table XXVII

Showing the main values of the Cytological Curves  
of the subjects of group (c).

<u>No.</u>	<u>Initials</u>	<u>Age at time of Investi- gation Years</u>	<u>Range of % of Cornified Cells dur- ing period of Investi- gation</u>	<u>Mean % of Cornified Cells dur- ing period of Investi- gation</u>	<u>Range of Deep Cells</u>
1c	Mrs. W.D.	40 1/12th	4-18%	7%	0-2%
2c	" S.D.	43 4/12ths	4-20%	7.8%	0-2%
3c	" M.R.	43	4-17%	6.9%	1-5%
4c	" D.R.	38 6/12ths	6-26%	11.3%	Nil
5c	" J.K.	41 5/12ths	5-31%	9.5%	0-2%
6c	" J.H.	40	4-23%	7.2%	0-1%
7c	" C.W.	48 7/12ths	3-26%	7.8%	0-2%
8c	" N.McG	49 2/12ths	3-31%	10.8%	0-5%
9c	" G.S.	39 6/12ths	6-26%	10.9%	Nil
10c	" B.D.	42 6/12ths	3-9%	4.8%	2-5%
11c	" E.J.	43 11/12ths	2-15%	7.3%	1-3%
12c	" S.M.	42 8/12ths	2-16%	6.1%	0-3%
13c	" K.R.	42 3/12ths	3-16%	5%	1-2%
14c	" C.L.	42 1/12th	4-30%	9.5%	0-2%

DISCUSSION

The estimation of oestrogenic activity by means of vaginal smears is a subject on which hundreds of papers have been published, and it is only necessary to read a few of them to appreciate that the results and conclusions of workers conducting comparable investigations differ widely.

This is, at least, partly due to their diverse interpretations of what constitutes cornification, and also to the fact that the criteria by which the various types of cell are to be differentiated varies greatly from author to author.

Absence of unanimity on these subjects and a diversity of method by which the cornification index is determined, renders difficult comparison between the results of one worker and another, and has prompted Murray and Osmond-Clarke (1956) in a recent issue of the British Medical Journal, to make a plea for the adoption of a standard method of estimating oestrogenic activity from vaginal smears.

In/

In this investigation it has therefore been considered necessary to define in detail a cornified, an intermediate and a deep cell, and the results have been reached on the basis of these definitions.

The values obtained for the cornification indices of the normal women of group (c), and also for those of the subjects of group (a) will be found to be generally much lower than those given by De Allende and Oriás in their book, "Cytology of the Human Vagina". While racial and climactic differences might be invoked to explain the disparity between their results and those obtained in this investigation, it is more probable that the essential difference arises from the question of what is to be regarded as a cornified cell and possibly even with what constitutes pyknosis.

In a personal communication (1955) and a practical demonstration, Dr. E. Wachtel of the Postgraduate School of Medicine, Hammersmith, indicated what were her standards of cornification, and stated that she had been unable to obtain from the vaginal smears of healthy ovulating British women the high percentages of cornified cells reported by de Allende and Oriás. Peak percentages, she/

she often found, did not exceed 30% and were sometimes considerably lower.

The actual numerical values obtained in this investigation are however of relatively minor interest. The essential fact is that, in the performance of the cell counts of all smears, a serious attempt has been made to maintain a constant standard of cellular differentiation, based on the definitions already stated.

This being so, the significance of the results becomes apparent when the graphs of the various subjects are studied and compared, and when the tabulated values obtained from the individual women are classified by groups.

The close similarity between the graphs and values of the subjects of groups (a) and (c) is then strikingly demonstrated, as is the sharp difference between the graphs and values of the subjects of both of these groups and those of group (b).

Although the range of percentage of cornified cells varies considerably from one subject to another, certain facts claim special attention.

The peak percentage values of the subjects of group (a) range from 8% to 40%, whereas among the subjects of group/



group (b) in no instance does the peak percentage of cornified cells exceed 4%. It is also to be noted that, although the lowest peak value for a subject of group (a) was obtained from a woman aged 51 (No. 23a), this value is twice as high as the highest returned by any woman of group (b).

An equally striking contrast is apparent when a comparison is made between the mean values for cornified cells, of the subjects of these two groups. Among the subjects of group (a) the mean values for cornified cells range from 4.6% to 19.1% while those of the subjects of group (b) range from zero to 1.4%.

Not only do the two ranges of values fail to overlap but, it is to be noted that in 13 of the 24 subjects of group (b) cell counts conducted on more than a dozen smears disclosed no cornified cells at all.

If the degree of cornification exhibited in the vaginal smear is to be considered as a quantitative index of the degree of oestrogenic activity of the subject, these results would imply that the minimum oestrogenic activity of any subject of group (a) substantially exceeded the maximum oestrogenic activity manifest by any subject of group (b).

It is not within the scope of this investigation to attempt a precise assessment of the degree of activity attributable to extraovarian sources of oestrogen. That such sources do exist has, for some years, been generally accepted. Engelhart (1930, 1932 and 1935), by demonstrating that crude lipid extracts of the adrenals possessed both oestrogenic and progestational properties when administered to castrated and immature animals, initiated a multiplicity of investigations, of which even the main ones cannot here be enumerated. Then followed the isolation of oestrone and progesterone from adrenal extracts by Beall (1939). In addition Kemp and Pedersen-Bjergaard (1937) discovered small amounts of oestrogen in the urine of oophorectomised women and Callow and Emmens (1940) obtained an oestrogenic response from the urine of each of 16 oophorectomised women though in some instances the extent of the response was very small.

Hadfield (1956) while observing that "oophorectomy and adrenalectomy temporarily abolish oestrogen production stress the fact that "after a period of months oestrogens "usually in low concentration again appear in the urine". He then states that the "tissue of origin of these "oestrogens/

"oestrogens is obscure", but that "it is possible that the retroperitoneal mesenchyme from which the ovaries and adrenal cortex are developed, retains its potentiality to produce oestrogens and may be responsible for their delayed production and slow appearance in the urine".

It is therefore possible that those cornified cells which were detected in the smears of some of the subjects of group (b) may be evidence of oestrogenic activity on the part of the adrenal or some other extragonadal source. The striking difference however between the results returned by the two groups (a) and (b) is such that, as has already been stated, the highest peak and mean values obtained from any subject of group (b) are still lower than the lowest peak and mean values obtained from any subject of group (a).

This disparity of values would suggest that conserved ovarian tissue, which for the purposes of this investigation, distinguishes the subjects of group (a) from those of group (b), is the source of the significantly greater oestrogenic activity that has been exhibited in the vaginal smears of the former group as compared with those of the latter.

That a similar investigation of the vaginal smears of the/

the subjects of group (b) conducted after an interval of a few more years might, in some cases, produce considerably higher values for cornified cells, was suggested by the fact that while this investigation was being undertaken, vaginal smears from two subjects, aged 67 and 69 years respectively, were also obtained, and it was observed that the degree of cornification exhibited in the smears of one of these women substantially exceeded that obtained from any subject of group (b) in this investigation, being in fact comparable to what was found in the smears of some of the subjects of group (a).

The comparatively low values obtained from the subjects of the oophorectomised group (b) may therefore be attributable to the fact that the interval between operation and investigation has been too brief to permit anything in the nature of large scale production of oestrogen by an extragonadal source. It must however be recalled that, in the case of one subject (4b) the interval between oophorectomy and investigation was 9 years.

When the graphs and tables of the subjects of group (a) are compared with those of the normal group (c), no significant/

significant differences are to be observed, for the peak percentages of cornified cells of the subjects of group (a) range from 8% to 40%, whereas those of the normal group (c) range from 9% to 31%.

Similarly, the mean percentages of cornified cells vary among the subjects of group (a) from 4.6% to 19.1%, whereas among the normal women of group (c) the variation is from 5% to 11.3%. The overlap is obvious and need not be stressed, and the similarity between the figures of Tables XXV and XXVI is sufficient to justify the supposition that the degree of oestrogenic activity to be found in the subjects of either of these groups is comparable to that exhibited by the other.

SUMMARY and CONCLUSIONS

Unlike the two previous investigations, which were essentially designed to ascertain whether or not ovaries conserved at hysterectomy continued to produce corpora lutea, this investigation was employed solely as an index of oestrogenic activity. No attempt was made to diagnose the presence or absence of ovulation by this method, although de Allende and Orias claim to be able to do so (1950D).

Since the menstrual cycles of women approaching the menopause are known to be not infrequently anovular, and as most of the subjects of this investigation were over 40 years of age, it seemed desirable that the oestrogenic properties of the conserved ovary should be studied as an independent activity.

For this reason, use was made of the fact that the vaginal epithelium is responsive to the stimulus of the ovarian hormones, and that by means of the vaginal smear, the desquamated epithelial cells may be made to serve as an index of the oestrogenic activity of the individual. Since oestrogen is essentially a growth hormone, and since the/

the desquamation of cornified cells is a consequence of pronounced vaginal epithelial hypertrophy, it follows that the percentage of cornified cells in a vaginal smear reflects the potency of the oestrogen of that particular subject.

Throughout this investigation the percentage of cornified vaginal epithelial cells has been employed as a quantitative index of the oestrogenic activity of the subject.

The histology of the vaginal epithelium is discussed, and the changes it undergoes at various stages during the lifetime of the human female are shown to be related to changes in the blood oestrogen levels.

Two attempts to assess ovarian oestrogenic activity following hysterectomy are reviewed briefly.

The former by de Allende and O'riás (1950C), though based on the results of only a few subjects, suggests that hysterectomy does not appear to exercise a harmful influence on the oestrogenic properties of the conserved ovary.

The latter by Bancroft-Livingston (1954), reaches a similar conclusion but may be criticised in that oophorectomised subjects were not used as controls.

The/

The method employed in this investigation is described in detail, and, the terms "deep cell", "intermediate cell" and "cornified cell" are defined, in order to indicate their meaning as used in this context.

Stress is laid upon the fact that the criterion of cornification is not an acidophylic cytoplasm but pyknosis of the nucleus.

Vaginal smears were obtained from the subjects of all three groups and the results of the differential cell counts performed are presented both graphically and in tabular form.

These results indicate a close similarity of values between the subjects of groups (a) and (c) i.e. between those women who had undergone hysterectomy with conservation of one or both ovaries and the normal subjects.

For example, among the subjects of group (a) the peak percentages of cornified cells ranged from 8% to 40% while the corresponding figures for the subjects of group (c) are 9% to 31%. On the other hand, the highest peak percentage of cornified cells to be observed in any subject of group (b) was 4%.

This trend is again in evidence when the mean values for/



for the percentages of cornified cells of all subjects in each group are studied.

Among the subjects of group (a) the mean percentages of cornified cells range from 4.6% to 19.1%, while the variation among the normal subjects is from 5% to 11.3%. When however, one considers the results of the castrated subjects of group (b), it is found that the smears obtained from 13 subjects of this group showed no cornified cells at all, and that the highest mean percentage of cornified cells to be obtained from a series of smears of any subject of group (b) was 1.4%.

The question of extragonadal sources of oestrogen is discussed and it is suggested that, were this investigation to be repeated on the same subjects in a few years' time, the values for cornified cells obtained from some of the subjects of group (b) might well be of a much higher order.

Reference is made to a parallel investigation of the vaginal smears of two elderly subjects aged 67 and 69 respectively, neither of whom had suffered from malignant disease, and it is noted that the percentages of cornified cells obtained from the smears of one of these two subjects greatly/

greatly exceeded the highest values to be observed in the smears of any subject of group (b).

While the relatively low percentages of cornified cells obtained from the smears of subjects of group (b) might be explained on the grounds that the interval between oophorectomy and investigation had not been sufficiently long to allow extragonadal sources to obtain maximum oestrogen production, it is emphasised that in one subject of this group (No. 4b), oophorectomy had occurred nine years before this investigation was undertaken.

If cornification of the vaginal epithelium is to be accepted as a quantitative index of the oestrogenic activity of the subject, it is clear from this investigation that the range of oestrogenic activity manifest among the subjects of group (a) is much greater than that which is to be observed among those of group (b), and is indeed comparable to what is to be found among the normal subjects of group (c).

Since, in these investigations, the factor which distinguishes the subjects of group (a) from those of group (b) is conserved ovarian tissue, it is justifiable to assume that the significantly higher percentages of cornified/

cornified cells to be found in the smears of the former group of women is an indication that their ovaries have continued to produce oestrogen although the uterus had previously been removed.



I N T R O D U C T I O N

This investigation is based upon the fact that the serum and urine of postmenopausal women are known to have a much greater content of pituitary gonadotrophin than is to be found in the serum and urine of the normal premenopausal woman. These relatively higher values are generally attributed to the progressive withdrawal of the inhibitory influence which is thought to be exercised by the high levels of the premenopausal ovarian oestrogen on the liberation of gonadotrophin by the pituitary (Bishop 1947).

Not unexpectedly, it has also been found that bilateral oophorectomy, when performed on premenopausal women, results in a similar sudden increase in urinary gonadotrophic values (Zondek 1932).

Heller Farney and Myers (1944), who conducted such an investigation on castrated women, reported their results as follows:

"All (patients) with normal preoperative levels showed a significant rise in gonadotrophic excretion as early as the sixth to tenth postoperative day. The menopausal zone was reached by the sixth day in 58 per cent, by the tenth day in 86 per cent and by the next assay conducted at the end of one month in all cases studied."

Estimations of the pituitary gonadotrophin in the urine of the subject being investigated, are conducted by the method of animal assay, and for this purpose a variety of animals have been used.

It was decided that, by means of such assays of the urinary gonadotrophin, an attempt should be made, in this investigation, to obtain an indirect assessment of the oestrogenic activity of those ovaries which had been conserved when hysterectomy was performed.

It was thought that if gonadotrophic values obtained from the urine of those women whose ovaries had been conserved when the uterus was removed, were found to be significantly lower than those obtained from the urine of that group of women from whom both uterus and ovaries had been removed, it might be considered as evidence that, in the former group, ovarian oestrogen still continued to hold the pituitary gonadotrophin in check, and could therefore be interpreted as a probable indication of the continuation of oestrogenic activity on the part of the conserved ovary.

#### Some Limitations of Gonadotrophic Assay

Investigations of urinary gonadotrophin are usually conducted/

conducted with either rats or mice as the test animals, and to obtain more accurate results these should be hypophysectomised, so that endogenous production of gonadotrophin from the pituitary of the test animal does not interfere with the results.

All modern attempts to assay the gonadotrophic hormones suffer from certain limitations.

The first limitation is the absence of an international standard with which unknown preparations can be compared. Results of assays are therefore usually expressed in arbitrary "rat" or "mouse" units which vary from one laboratory to another.

Secondly, in such investigations, it is probable that at least two distinct substances are being estimated simultaneously, and these may be present in different concentrations at different times.

Then the quantities of gonadotrophin in the selected samples of urine are often very small, necessitating elaborate methods of preparation and concentration. These procedures, according to Loraine (1952), may sometimes produce a loss of gonadotrophic activity and result in the production of extracts which prove toxic to the experimental animal.

The/

The method employed in the present investigation makes use of neither the rat nor the mouse as the experimental animal, but of the male toad (*Xenopus laevis*).

This is not due to a belief in the superiority of this method of assay but rather to the fact that circumstances precluded the adoption of any of the other methods which are considered to be more sensitive and accurate.

The Nature of the Gonadotrophic Hormone in the Urine of the Castrated and Menopausal Subject

The preparation, by a number of workers, for example Fevold, Hisaw and Leonard (1931), Wallen-Lawrence (1934), Loeb Saxton and Hayward (1936), and Dadds and Noble (1936) of pituitary extracts which appear to exhibit either an almost purely follicle-ripening effect or an almost purely interstitial cell stimulating effect, has led to a generally accepted subdivision of pituitary gonadotrophin into (1) the Follicle Stimulating (or Ripening) Hormone, i.e. FSH, and (2) the Interstitial Cell Stimulating Hormone, i.e. ICSH. The latter was formerly known as the Luteinising Hormone or LH, but since it can be assayed in the male animal the term Interstitial Cell Stimulating Hormone is now preferred.

It/



It is necessary, however, to recall that a complete separation of FSH and ICSH has not yet been achieved with certainty.

The gonadotrophin which is to be found in the urine of the castrated or postmenopausal woman is usually believed to have a predominantly follicle-ripening action (Bishop 1947), Katzman and Doisy (1934)), yet Frank, Salmon and Friedman (1935) and Salmon and Frank (1936) have reported that while this substance was found by them to produce such an effect on infantile rats, an increase in dose achieved a "luteinising" effect in the same animals.

Then Loraine (1952), writing on "Recent Developments in the Clinical Application of Hormone Assay", refers to a personal communication from McArthur in which the latter, as a result of preliminary studies concludes that "menopausal women excrete both FSH and LH" (i.e. ICSH).

McArthur's assay made use of the rat as the test animal, the potency of the ICSH in the subject's urine being determined by its capacity to produce enlargement of the ventral lobe of the rat prostate.

The method of assay adopted in the present investigation possessed the advantage of measuring both FSH and ICSH and so may be considered to reflect the potency of the subject's total gonadotrophin.

PREVIOUS INVESTIGATIONS

A prolonged and diligent search of the relevant literature disclosed remarkably few attempts to investigate the functional state of the conserved ovary by animal assay of the pituitary gonadotrophin, and no recent work on this subject was discovered.

Tamis (1934), in a study of the relationship between menopausal symptoms and ovarian function following hysterectomy, conducted a series of biological assays of F.S.H. by injecting extracts of the urine of his subjects into immature mice, and observing whether or not the injected material was capable of producing hypertrophy of the mouse ovary. The hypertrophy, which when present was attributed to enlargement of the primordial follicles, was considered to constitute a positive sign of the presence of F.S.H. in the urine. The results were not expressed in quantitative terms and were not measured against any standard preparation, but were considered in conjunction with another biological assay of the urinary oestrogen of the subject being investigated.

Tamis/

Tamis, commenting on his results, concludes that the duration of ovarian function following hysterectomy is not solely related to the amount of gonadal tissue conserved, and he ends by making a plea for maximal conservation of uterine mucosa, on the supposition that the severity of the menopausal symptoms is directly related to the amount of endometrium removed.

Marx, Catchpole and McKennon (1936) in an investigation of the same subject, conducted in a similar fashion, also reached the conclusion that

"the preservation of even a small part of the uterus "seems to have a retarding and mitigating effect upon the "appearance of retrogressive changes in the pituitary- "ovarian function and the occurrence of menopausal symptoms."

In the reports of both of these investigations, the authors therefore stress the importance of endometrial conservation, without which ovarian preservation is thought to be of relatively little account.

In 1937 Krane published the results of a number of assays of pituitary gonadotrophin. The subjects of his investigations were patients suffering from various gynaecological disorders, and among them were some who had undergone the operation of hysterectomy with conservation of/  
of/

of one or both ovaries. The urine of such women he injected into immature mice, adopting a modified form of the technique originally described by Zondek (1930).

The results of these assays were presented in terms of units of a standard preparation, and the conclusion reached by Krane was that premenopausal women who have had the uterus removed with conservation of one or both ovaries, do not return higher values for pituitary gonadotrophin than do normal women. Unfortunately, the results were not compared with those of a control group whose ovaries had been removed, but it is surprising that this work seems to have passed almost unrecognised, as more recent literature is conspicuously lacking in any attempt to confirm or refute its conclusions.

#### M E T H O D

The subjects of this investigation consisted of women of groups (a) and (b). All therefore had undergone the operation of hysterectomy, some (i.e. those of group (a)) having had one or both ovaries conserved, while others (those/

(those of group (b)) had had them removed.

The co-operation of 20 of the 24 women (i.e. 83.3%) of each of these groups was obtained.

The original plan included the performance of assays of the urine of the subjects of all three groups. It is known, however, that the gonadotrophin values obtained from the urine of normal premenopausal women vary widely according to the different phases of the menstrual cycle, peak values being reached about the time of ovulation. At other stages of the cycle the values are frequently so low that bio-assay is sometimes not practicable, unless a further concentration of the urine is made. Temperature records were therefore used in an attempt to anticipate that stage in the menstrual cycle at which measureable values might be expected. Unfortunately, the temperature records proved a very uncertain guide, and as the submission of a large number of samples of urine from each woman of group (c) was impracticable, it was decided in this investigation to assay only samples of urine from the subjects of groups (a) and (b).

Two 24-hour samples of urine (usually separated by an interval of one week), were obtained from each of the forty subjects/

subjects. These samples of urine were concentrated by the Kaolin method so that each ml. of the concentrate was equivalent to 12 ml. of urine (Scott 1940).

The concentrates were assayed by the method which has been described by Hobson (1952) and also by Hobson and Landgrebe (1954).

The following is a brief account of the technique employed.

Groups of male toads (*Xenopus laevis*) were injected with concentrate and thereafter placed in separate jars. The injections were made into the dorsal lymph sac.

Four hours later, a few drops of urine were collected with a pipette from the bladder of the toad. The urine thus obtained was placed upon a slide and examined microscopically for spermatazoa, the appearance of which constitutes a positive reaction.

A clean pipette was used for each animal in order to avoid contamination. Any animal under test, which gave a negative reaction at four hours, was returned to its jar and re-examined 24 hours after injection.

The results of these assays were expressed in units of H.M.G.20A. This material was prepared by Messrs. Organon Laboratories/

Laboratories from the urine of postmenopausal women. The groups of male toads used in this investigation were standardised against this preparation. Dose response curves were prepared and it was found that the \*MED50 for this powder was equivalent to 1.2 mg. of H.M.G.20A. This was called one unit.

\* MED50 signifies the mean effective dose which achieves a 50% response.

RESULTSTable No. XXVIII

Showing results of biological assays of the urine of 20 women of group (b), i.e. subjects who had undergone hysterectomy with bilateral oophorectomy.

<u>No.</u>	<u>Initials</u>	<u>Age at time of Investi- gation Yrs.</u>	<u>Interval since Hyst. Yrs.</u>	<u>Type of Hyst. Total or Subtotal</u>	<u>Units of HMG.20A per 24-hours (Mean Value of two 24-hour samples of urine)</u>
1b	Mrs. A.S.	43	2 7/12ths	Total	309 units
2b	" A.Y.	44 11/12ths	2	"	483 "
3b	" A.C.	47	3 6/12ths	"	118 "
4b	" M.W.(C)	40 10/12ths	10 5/12ths	Subtotal	447 "
5b	" H.W.	48 11/12ths	3 5/12ths	Total	200 "
6b	Miss B.F.	50 1/12th	2 5/12ths	"	194 "
7b	Mrs. M.B.	43 7/12ths	2 8/12ths	"	264 "
8b	" J.S.	49 10/12ths	3 3/12ths	"	159 "
9b	" M.S.(C)	51 6/12ths	2	"	219 "
10b	" A.H.	41 1/12th	2 2/12ths	"	187 "
11b	" M.S.(K)	45 3/12ths	2 1/12th	"	420 "
12b	" C.C'G	42 6/12ths	3 8/12ths	"	472 "
13b	" M.P.	43 6/12ths	4 4/12ths	Subtotal	275 "
14b	" P.F.	31 10/12ths	2 10/12ths	Total	552 "
15b	" A.T.	35 4/12ths	3 7/12ths	Subtotal	441 "
16b	" M.D.	43 2/12ths	2 10/12ths	"	389 "
17b	" C.P.	49 6/12ths	2 9/12ths	"	182 "
18b	" M.W.(D)	44 4/12ths	2 5/12ths	Total	287 "
19b	" E.M.	46 5/12ths	2 5/12ths	Subtotal	189 "
20b	" A.W.	48	3	Total	251 "

Range of Values of this group = 118 to 552 units HMG.20A

Mean Value for this group = 301.9 units HMG.20A

Ages range from 31 10/12ths to 51 6/12ths years

average age of group at time of investigation = 44.35 years

Range of interval since hysterectomy = 2 years to 10 5/12ths years

average interval since hysterectomy = 3.2 years



Table No. XXIX

Showing results of biological assay of the urine of 15 women of group (a) i.e. subjects who had undergone the operation of hysterectomy with conservation of one or both ovaries.

No.	<u>Initials</u>	<u>Age at time of Investigation Yrs.</u>	<u>Interval since Hyst. Yrs.</u>	<u>Type of Hyst. Total or Subtotal</u>	<u>No. of Ovaries Conserved</u>	<u>Units of HMG. 20A per 24-hours (Mean Value of two 24-hour samples of urine)</u>
a	Mrs. B.D.	42	4 6/12ths	Total	Both	34 units
a	" M.M.	43 8/12ths	2 5/12ths	"	"	26 "
a	" J.C.(K)	43 11/12ths	4	"	"	49 "
a	" M.McD	44 6/12ths	4	Subtotal	"	52 "
a	" J.C.(B)	38	3 10/12ths	Total	"	30 "
a	" M.H.(C)	38 6/12ths	3 7/12ths	"	"	31 "
10a	" H.T.	41 6/12ths	2 9/12ths	"	"	43 "
11a	" C.K.	39 11/12ths	2 6/12ths	Subtotal	"	50 "
12a	" A.R.	47 8/12ths	2 10/12ths	Total	"	41 "
13a	" M.H.(T)	42 5/12ths	7 10/12ths	Subtotal	"	47 "
14a	" J.S.	41	3 4/12ths	Total	"	34 "
15a	" I.R.	46 5/12ths	2 11/12ths	Subtotal	"	23 "
18a	" H.P.	47 1/12th	2 8/12ths	Total	One	54 "
19a	" E.C.	47 1/12th	2 11/12ths	Subtotal	One	42 "
20a	" J.O'N	41	2	"	One	63 "

Ages of these 15 women of group (a) range from 38 to 47 8/12ths years  
 Average age of these 15 subjects of group (a) = 42.9 years  
 Range of interval since Hysterectomy = 2 years to 7 10/12ths years  
 Average interval since Hysterectomy = 3.47 years  
 Range of Values of these 15 women of group (a) = 23 to 63 units HMG.20A  
 Mean Value for these 15 subjects = 41.26 units HMG.20A.

The values obtained from the urines of the remaining five subjects of group (a) who took part in this investigation have been separately tabulated, as all five had complained of recent attacks of "hot flushes." In the case of four of them/

them (Nos. 3a, 4a, 5a and 16a) this symptom had originated during the year preceding this investigation, i.e. several months after the previous four investigations had been completed. Although the other subject (No. 21a) had previously complained of hot flushes immediately following the operation of hysterectomy, they were said, by the patient, to have ceased within a few weeks (See "Discussion" under "First Investigation"). It is significant that, at the time when this fifth and final investigation was being undertaken, she stated that there had been a recent recurrence of "hot flushes" in a much more severe form.

Moreover, these five subjects had each by the time this investigation was being conducted, attained the age of 48 years. It has been assumed therefore, that they had reached the menopause and for this reason the results obtained from them have been separated from those of the larger (and younger) group.

Table No. XXX

Showing results of biological assay of the urine of 5 women of group (a) who, by reason of age and recent vasomotor symptoms were assumed to have reached the menopause.

<u>No.</u>	<u>Initials</u>	<u>Age at time of Investi- gation Yrs.</u>	<u>Interval since Hyst. Yrs.</u>	<u>Type of Hyst. Total or Sub total</u>	<u>No. of Ovaries Con- served</u>	<u>Units of HMG. 20A per 24- hours (Mean Value of two 24-hour samples of urine)</u>
1a	Mrs. J.F.(1)	50 6/12ths	3 7/12ths	Total	Both	910 units
1a	" E.D.	48 1/12th	2 10/12ths	"	"	208 "
5a	" J.F.(B)	48 1/12th	3 9/12ths	"	"	399 "
16a	" J.McI	49 6/12ths	2 6/12ths	"	One	488 "
21a	" M.D.	49 8/12ths	2 7/12ths	"	One	201 "

Ages range from 48 1/12th to 50 6/12ths years

Average age of these five women of group (a) = 49.17 years

Range of interval since Hysterectomy = 2 6/12ths years to 3 9/12ths years

Average interval since Hysterectomy = 3.05 years

Range of Values of these five subjects of group (a) = 201 units to 910 units HMG.20A

Mean Value of these five subjects of group (a) = 433.2 units HMG.20A

DISCUSSION

The decision to perform biological assays of the urine of the five subjects of group (a) whose results have been separately tabulated, was taken in the knowledge that they might well return values differing widely from those of the other fifteen. The possibility that any subject of group (a) might reach a natural menopause was always present during these investigations, and that possibility was particularly strong among those subjects who were approaching, or had attained, the age of 47, the average age, according to Wilfred Shaw (1943) at which women of this country experience the onset of the change of life.

The fact therefore, that five women of this group, all of whom had passed the age of 48 years, should complain of recent vasomotor symptoms, is not one that should occasion surprise or demand special explanation, nor is there any reason why this symptom, occurring about the time when a physiological menopause is to be expected, should be associated with an operation which the patient had undergone some years earlier.

A/

A more just assessment of the effect of hysterectomy is to be obtained by comparing the results of the younger women, (i.e. those under 48 years of age) of group (a), with those of the oophorectomised subjects of group (b). If, therefore, one excludes the five subjects over 48 years of age, and considers the subgroup consisting of the remaining 15 subjects of group (a) whose ages ranged from 38 years to 47 years 8 months (with an average of 42.9 years), there is, at least, less likelihood that the conclusions will be disturbed by the fortuitous influence of an incidental physiological menopause, and greater opportunity will thus be afforded for an evaluation of the effect exerted by the operation of hysterectomy on ovarian function.

Even within this subgroup, however, the influence of the normal menopause might have been expected to show itself, since the group contains three women over 47 years of age, and a physiological menopause at that age and indeed at ages younger than that, is by no means uncommon.

The first significant feature of these results is therefore the consistently low values obtained from the urine of those 15 women of group (a) whom we may designate as the under-48 subgroup. The values range from 23 to 63 units/

units HMG.20A, the mean figure for the group being 41.26 units. When these figures are compared with those of the oophorectomised subjects of group (b), the contrast between the two sets of results becomes immediately apparent. The 20 subjects of group (b) whose ages ranged from 31 years 10 months to 51 years 6 months, with a group average of 44.35 years, produced values which ranged from 118 units HMG.20A to 552 units HMG.20A with a group mean value of 301.9 units. In other words, the lowest value obtained from a subject of group (b) (118 units) is approximately twice as high as the highest value (63 units) obtained from any of the 15 subjects of group (a) who had not, at the time of investigation, attained the age of 48 years of age. It is also significant that the mean value for group (b) (301.9 units) is many times higher than the mean value for the 15 subjects of group (a) who form this under-48 subgroup (41.26 units).

The results obtained from the 5 women of group (a) who, at the time of investigation were each over 48 years of age, and who all complained of recent attacks of hot flushes, reflects a close relationship with those returned by the castrated women of group (b). In this small subgroup the range of values is from 201 to 910 units HMG.20A with a mean value/

value of 433.2 units. The statistics of the assay in fact indicate an affinity between these subjects and the women of group (b), an affinity confirmed by symptomatology and leading to the assumption that the five women of this subgroup had, at the time of this investigation, entered upon the menopause.

Series as small as these do not provide the ideal statistical basis for pronouncements on the relative probability of continued ovarian activity following subtotal as distinct from total hysterectomy. One glance at Table No. XXIX however, will reveal the fact that the range of values for pituitary gonadotrophin obtained from the subjects who had undergone the operation of subtotal hysterectomy do not appear to differ significantly from those obtained from subjects who had undergone total hysterectomy.

Moreover, as the same table will indicate, the range of values obtained from women under the age of 48 years of age, who had both ovaries conserved does not differ widely from the range of values obtained from those women who had one ovary removed.

SUMMARY and CONCLUSIONS

This investigation depends upon the fact that the urine and serum of postmenopausal women have a greater content of pituitary gonadotrophin than the urine and serum of premenopausal women. The higher values are generally attributed to the withdrawal of the inhibitory influence thought to be exercised on the gonadotrophin of the pituitary by the high oestrogen output of the premenopausal ovary (Bishop 1947).

It is possible that future research may show the above explanation to be an oversimplification of what may prove to be a more complex process, but there is little doubt that the physiological menopause, like surgical castration, is followed by a pronounced and sustained rise in the values to be obtained for the serum and urinary gonadotrophin of the menopausal or castrated subject.

It would appear logical therefore, to assume that if (as is believed by some), ovaries which have been conserved at hysterectomy soon cease to function, the consequent fall in the oestrogen output should produce as one of its sequelae  
a/



a marked rise in the urinary gonadotrophic values of the hysterectomised subject.

The object of this investigation was therefore to discover whether such significant increases in pituitary gonadotrophic values could be observed in the urine of those women who had undergone the operation of hysterectomy with partial or complete conservation of the ovaries, and to compare the values obtained from this group of women with those obtained from the oophorectomised subjects of group (b).

The method used to detect pituitary gonadotrophin in the urine of the subject is that of animal assay, and the limitations of this method are discussed critically.

The preparation of pituitary extracts which exhibit either an almost purely follicle-ripening effect or an almost purely interstitial cell stimulating effect, has led to a generally accepted subdivision of pituitary gonadotrophin into (1) the Follicle Ripening Hormone (FRH) and (2) the Interstitial Cell Stimulating Hormone (ICSH), though it must be remembered that a complete separation of FRH and ICSH has not yet been achieved.

Although the gonadotrophin which is found in the urine/

urine of castrated and postmenopausal women is generally thought to have a predominantly follicle ripening action, evidence is quoted to indicate that it also possesses a luteinising effect. Since the method of assay adopted in this investigation is employed both in the estimation of FRH and ICSH, it may be considered to measure the subjects' total gonadotrophin.

Previous investigations of the pituitary gonadotrophin of women who had undergone hysterectomy are discussed.

Tamis (1934) and Marx, Catchpole and McKennon (1936) performed assays of the pituitary gonadotrophin of hysterectomised women by injecting extracts of the subjects' urine into immature mice and observing whether or not the injected material was capable of producing hypertrophy of the mouse ovary.

The results were, in neither case, expressed in quantitative terms, nor were they measured against a standard preparation.

Both investigations seemed to indicate that while ovarian activity may persist after hysterectomy, the possibility is enhanced, if some endometrial tissue is preserved.

Krane/

Krane (1937) whose method of assay was a modification of that originally described by Zondek (1930), reported that women who have had the uterus removed with conservation of ovaries, do not return higher values for pituitary gonadotrophin than do normal women.

Originally it was intended that, in this investigation, all three groups of subject should participate, but as this was found to be impracticable, it was decided to invite as many as possible of the subjects of groups (a) and (b) to assist in this final assessment of ovarian activity following hysterectomy.

Twenty of the twenty-four subjects of group (b) (i.e. women who, some years earlier, had undergone the operation of hysterectomy with bilateral oophorectomy), whose ages ranged from 31 years 8 months to 50 years 6 months, took part in this investigation. Each submitted two samples of urine which were assayed for pituitary gonadotrophin by the method described by Hobson (1952) and by Hobson and Landgrebe (1954), the test animal being the male toad (*Xenopus laevis*).

The results, expressed in units of HMG.20A, showed that the values for this group of women ranged from 118 to

552 units HMG.20A with a mean value for the group of 301.9 units.

Twenty of the twenty-four subjects of group (a) (i.e. women who, some years earlier, had undergone the operation of hysterectomy with conservation of one or both ovaries) also took part in the investigation. These have been divided into two subgroups.

One subgroup consisted of 5 subjects, all of whom were over 48 years of age and each of whom had recently begun to experience vasomotor symptoms. Because of the recent onset of these symptoms, and in view of the fact that a physiological menopause is to be expected at this age, these subjects were regarded as probably menopausal, and have been considered as distinct from the main group.

The ages of this subgroup ranged from 48 years 1 month to 50 years 6 months. The biological assays (of pituitary gonadotrophin) of the urine of the subjects of this subgroup produced values which ranged from 201 units to 910 units HMG.20A, with an average value for the subgroup of 433.2 units.

The remaining subgroup of group (a) consisted of 15 women, all of whom were under 48 years of age at the time when/

when this investigation was conducted, and none of whom gave a history of recent onset of vasomotor symptoms. Their ages ranged from 38 years to 47 years 8 months. Bio-assay of the urine of this subgroup for pituitary gonadotrophin produced values ranging from 23 to 63 units HMG.20A with a mean value for the subgroup of 41.26 units.

If one excludes from consideration the subgroup consisting of those subjects who, on the basis of subjective evidence and age may be assumed probably to have reached a menopause that cannot be described as premature, in terms of the average age of onset of a physiological menopause, one is left with a group of women from whom one is then more able to form an estimate of the effect of hysterectomy on the function of the conserved ovary.

Since the highest value for pituitary gonadotrophin (63 units HMG.20A) obtained from the urine of a subject of the remaining subgroup of 15 women, is much lower than the lowest value (119 units HMG.20A) obtained from the urine of a woman whose uterus and ovaries had both been removed, one is led to conclude that some common factor probably operated in the subjects of this subgroup ensuring a range of values of a different order from both those obtained from/

Table No. XXXI

in which an estimation of the ovarian activity of each subject of group (a) is made on the basis of the results of all investigations.

No.	Initials	Menopausal Index (age in brackets) Years	Vasomotor Symptoms	Pattern of Temperature Record (age in brackets) Years	Classification of Subject on basis of Temperature Record + Pregnanediol Estimations	Range of % of Cornified Cells with Mean Values in brackets	Results of Pituitary Gonadotrophin Esti- mations, Mean Values of two 24-hour samples of urine (age in brackets) Years	Probable State of Ovarian Activity (on basis of Results of all Investigations)
1a	Mrs. B.D.	0 (40 6/12ths)	Nil	Diphasic (40 6/12ths)	Prob. Ovulating	7-23% (11.7%)	34 units (42)	Oestrogenic activity present. Poss. ovulating.
2a	" M.M.	14 (42 8/12ths)	Do.	Do. (42 5/12ths)	Do.	6-40% (15.3%)	26 " (43 8/12ths)	Do.
3a	" J.F.(D)	2 (48 6/12ths)	Do.	Monophasic (48 4/12ths)	Prob. Not ovulating.	4-11% (6.5%)	910 " (50 6/12ths)	Menopause prob. reached during investigations.
4a	" E.D.	0 (46 3/12ths)	Do.	Do. (46 9/12ths)	Do.	5-14% (7.2%)	208 " (48 1/12th)	Do.
5a	" J.F.(B)	11 (45 10/12ths)	Do.	Do. (46 2/12ths)	Do.	4-11% (5.6%)	399 " (48 1/12th)	Do.
6a	" J.C.(K)	1 (42 9/12ths)	Do.	Prob.Diphasic (42 9/12ths)	Prob.ovulating	8-37% (17%)	49 " (43 11/12ths)	Oestrogenic activity present. Poss. ovulating.
7a	" M.McD	3 (43 6/12ths)	Do.	Diphasic (43 6/12ths)	Do.	7-16% (9.1%)	52 " (44 6/12ths)	Do.
8a	" J.C.(B)	2 (36 6/12ths)	Do.	Do. (36 8/12ths)	Do.	8-22% (10.2%)	30 " (38)	Do.
9a	" M.H.(C)	0 (37 1/12th)	Do.	Do. (37 1/12th)	Do.	15-28% (19.1%)	31 " (38 6/12ths)	Do.
10a	" H.T.	12 (40 3/12ths)	Do.	Do. (40 9/12ths)	Poss.ovulating	8-19% (9.5%)	43 " (41 6/12ths)	Do.
11a	" C.K.	3 (38 5/12ths)	Do.	Prob.Diphasic (39)	Do.	5-16% (8.7%)	50 " (39 11/12ths)	Do.
12a	" A.R.	8 (46 5/12ths)	Do.	Diphasic (47 3/12ths)	Do.	4-17% (7.1%)	41 " (47 8/12ths)	Do.
13a	" M.H.(T)	14 (41 1/12th)	Mild	Do. (41 1/12th)	Prob.ovulating	8-23% (12.4%)	47 " (42 5/12ths)	Do.
14a	" J.S.	4 (39 5/12ths)	Nil	Monophasic (39 6/12ths)	Prob.not ovulating	6-15% (7.7%)	34 " (41)	Do.
15a	" I.R.	6 (45 1/12th)	Do.	Diphasic (45 1/12th)	Results inconclusive	4-14% (7.1%)	23 " (46 5/12ths)	Oestrogenic activity present.
16a	" J.McI	14 (48)	Do.	Prob.Diphasic (48 8/12ths)	Poss.ovulating	5-17% (8%)	488 " (49 6/12ths)	Menopause prob.reached during investigations.
17a	" M.W.	4 (33 2/12ths)	Do.	Diphasic (33 6/12ths)	Prob.ovulating	8-17% (11.7%)	Not per formed	- Oestrogenic activity present. Poss.ovulating
18a	" H.P.	2 (45 8/12ths)	Do.	Do. (46)	Poss.ovulating	8-20% (8.7%)	54 " (47 1/12th)	Do.
19a	" E.C.	0 (46)	Do.	Do. (46 7/12ths)	Poss.ovulating	4-19% (8.1%)	42 " (47 1/12th)	Do.
20a	" J.O'N	0 (40)	No.	Prob.Diphasic (40 4/12ths)	Poss.ovulating	8-20% (11.4%)	63 " (41)	Do.
21a	" M.D.	14 (48 4/12ths)	Do.	Do. (48 8/12ths)	Prob.ovulating	4-12% (7.5%)	201 " (49 8/12ths)	Menopause prob.reached during investigations.
22a	" J.B.	14 (40 2/12ths)	Moderate	Monophasic (40 9/12ths)	Prob. not ovulating	5-29% (11.5%)	Not per formed	- Oestrogenic activity present.
23a Miss	M.S.	4 (51)	Mild	Do. (51)	Do.	3-8% (4.6%)	Do.	- Probably at menopause.
24a Mrs.	M.C.	13 (41 3/12ths)	Moderate	Do. (41 9/12ths)	Do.	7-19% (8.9%)	Do.	- Oestrogenic activity present.

Group Average Menopausal Index of group (b) (oophorectomised subjects) = 20.42  
 " " " (c) (normal subjects) = 2.36  
 Group Mean % of Cornified Cells of Normal Group (c) = 7.9%  
 Pituitary Gonadotrophin Estimation Mean Value for the 20 castrated  
 subjects of group (b) = 301.9 units.

from the subjects who, by age and symptomatology, were believed to be menopausal.

In these circumstances, it is not unreasonable to conclude that the common factor was ovarian tissue which, at the time of investigation, continued to produce oestrogen.

W.C.	41 3/12ths		1 1/12ths
H.(D)	47 6/12ths		1 1/12ths
M.	48 1/12th	"	2 10/12ths
H.(B)	49 1/12th	"	3 9/12ths
H.(X)	43 11/12ths	"	4
W.C.D	46 5/12ths	Subtotal	4
H.(A)	47	Total	1 10/12ths
H.(D)	47 3/12ths	"	3 7/12ths
T.	48 6/12ths	"	2 1/12th
K.	46 11/12ths	Subtotal	2 6/12ths
R.	47 8/12ths	Total	2 10/12ths
H.(T)	48 3/12ths	Subtotal	1 10/12ths
S.	47	Total	1 2/12ths

Table XXXII

showing the probable state of ovarian activity of each subject of group (a) at the conclusion of all investigations. The Table also shows the age of each patient, the type of hysterectomy performed in each case, and the interval that elapsed between the performance of the operation and the final investigation.

<u>No.</u>	<u>Initials</u>	<u>Age at Conclusion of all Investigations Years</u>	<u>Type of Hysterectomy</u>	<u>Interval since Hysterectomy at conclusion of final investigation Years</u>	<u>Probable State of Ovarian Activity (on basis of Results of all Investigations)</u>
1a	Mrs. B.D.	42	Total	4 6/12ths	Oestrogenic activity present. Poss.ovulating
2a	" M.M.	43 8/12ths	"	2 5/12ths	Do.
3a	" J.F.(D)	50 6/12ths	"	3 7/12ths	Menopause prob. reached dur. investigations
4a	" E.D.	48 1/12th	"	2 10/12ths	Do.
5a	" J.F.(B)	48 1/12th	"	3 9/12ths	Do.
6a	" J.C.(K)	43 11/12ths	"	4	Oestrogenic activity present. Poss.ovulating.
7a	" M.McD	44 6/12ths	Subtotal	4	Do.
8a	" J.C.(B)	38	Total	3 10/12ths	Do.
9a	" M.H.(C)	38 6/12ths	"	3 7/12ths	Do.
10a	" H.T.	41 6/12ths	"	2 9/12ths	Do.
11a	" C.K.	39 11/12ths	Subtotal	2 6/12ths	Do.
12a	" A.R.	47 8/12ths	Total	2 10/12ths	Do.
13a	" M.H.(T)	42 5/12ths	Subtotal	7 10/12ths	Do.
14a	" J.S.	41	Total	3 4/12ths	Do.
15a	" I.R.	46 5/12ths	Subtotal	2 11/12ths	Oestrogenic activity present. Menopause prob. reached dur. investigations.
16a	" J.McI	49 6/12ths	Total	2 6/12ths	Oestrogenic activity present. Menopause prob. reached dur. investigations.
*17a	" M.W.	33 6/12ths	Subtotal	1 5/12ths	Oestrogenic activity present. Poss.ovulating



<u>No.</u>	<u>Initials</u>	<u>Age at Conclusion of all Investi- gations Years</u>	<u>Type of Hyster- ectomy</u>	<u>Interval since Hyster- ectomy at conclusion of final Investi- gation Years</u>	<u>Probable State of Ovarian Activity (on basis of Results of all Investi- gations)</u>
18a	Mrs. H.P.	47 1/12th	Total	2 8/12ths	Oestrogenic activity present. Poss. ovulating.
19a	" E.C.	47 1/12th	Subtotal	2 11/12ths	Do.
20a	" J. O'N	41	"	2	Do.
21a	" M.D.	49 8/12ths	Total	2 7/12ths	Menopause prob. reached dur. investigations.
*22a	" J.B.	42 9/12ths	"	5 6/12ths	Oestrogenic activity present.
*23a	Miss M.S.	51	"	1 10/12ths	Prob. at menopause.
*24a	Mrs. M.C.	41 9/12ths	Subtotal	7	Oestrogenic activity present.

\* Subjects marked by an asterisk were not investigated by the Fifth Method (i.e. Pituitary Gonadotrophin Estimation of Urine).

## DISCUSSION OF THE RESULTS OF ALL INVESTIGATIONS

When, at the conclusion of these investigations, an attempt is made to assess the probable state of the ovarian activity of each individual in group (a), two considerations must be borne in mind.

In the first place, it was unfortunately not possible to investigate every subject by all the methods employed. No estimation of the pituitary gonadotrophin of the urine of subjects 17a, 22a, 23a and 24a was performed. Assessment of the ovarian activity of these women must therefore be made on the results of the four earlier tests. The remaining twenty subjects were however investigated by all five methods.

Secondly, it must be remembered that the performance of the various tests covered a period of more than two and a half years. This means that care must be taken to ensure that what appeared to be a probability twelve months before the investigations ended, is not claimed as such a year later.

### Continuance of Oestrogenic Activity

The two investigations specifically designed to supply information/

information concerning the oestrogen producing powers of the conserved ovaries of group (a) subjects were (1) that in which the vaginal smears were examined for evidence of cornification, and (2) that in which the urine was estimated quantitatively by animal assay, for pituitary gonadotrophin. It is on the results of these tests therefore that an evaluation of the oestrogenic properties of the ovaries of the various subjects must be made.

(1) The Vaginal Smear

Attempts to classify vaginal smears according to the degree of oestrogenic activity which they are thought to exhibit, are usually somewhat unsatisfactory.

The literature on this subject abounds in articles in which smears are described as showing "mild" or "moderate" oestrogenic activity, without the reader being made aware of the author's criteria of demarcation. There is often in consequence, a regrettable lack of precision concerning the standards by which smears have been placed in one category or another.

Anyone who has conducted an investigation on the vaginal smears/

smears of a number of apparently normal women will concede that the range of percentage of cornified cells to be found in the smears of one woman during a complete menstrual cycle, may differ greatly from that to be found in the smears of another apparently normal woman during a similar period of time.

The problem is made no easier by the variety of methods which have been used to measure cornification curves, and by a diversity of opinion on what constitutes a cornified cell.

To avoid a retreat into nebulous terminology an attempt has been made to express the results of the fourth investigation, as far as possible, in mathematical form, and in the hope of rendering discussion more lucid, the terms "cornified cell", "intermediate cell" and "deep cell" have been defined to indicate the scope of their application in the present context.

In this investigation it was also decided that the mean percentage of cornified cells exhibited in the smears of each subject over a period of four weeks should be determined, and that this figure should be used as indicative of the degree of oestrogenic activity of the ovaries of that particular woman.

This device, though imperfect, possesses the merit of attempting/

attempting to correlate the degrees of cornification shown in the smears of several different subjects, on a basis that is immediately comprehensible in quantitative terms.

It was obviously desirable to relate these figures to a yardstick of normality and for this purpose a calculation was made of the mean percentage of cornified cells to be found in all the smears of all the subjects of group (c), vaginal smears having been taken from this group of normal women daily throughout one menstrual cycle. The value obtained from this calculation was 7.9%, a figure which is probably lower than might have been expected, especially when compared with some of the mean values (for percentages of cornified cells) returned by some of the subjects of group (a).

It was felt that if the mean percentage of cornified cells to be found during one menstrual cycle, in the smears of 14 normal women, is 7.9%, then it is probably justifiable to assume that any subject of group (a), whose mean percentage value for cornified cells (based on the results of 28 consecutive smears) exceeds this figure, probably possesses ovaries of oestrogenic potency equal to that of a normal woman of comparable age.

Reference/

Reference to Table XXV will show that of the 24 women of group (a) who had undergone hysterectomy with conservation of one or both ovaries no fewer than 16 showed mean values (for percentages of cornified cells) that exceeded 7.9%. It is assumed therefore that the oestrogenic activity of the ovaries of these subjects was equal to that of a normal woman of comparable age who had undergone neither hysterectomy nor oophorectomy.

Moreover, since some of the normal subjects of group (a) who, on the evidence of premenstrual endometrial biopsy, were known to be still ovulating, returned values as low as 7% (No. 2c) and 6.9% (No. 3c), it is probably justifiable to credit any woman of group (a) whose mean value exceeded 7% as possessing oestrogenic powers which can be classified as within normal limits. By this criterion 21 of the 24 subjects of group (a) may be said to exhibit, in their vaginal smears, the evidence of normal oestrogenic activity.

It is interesting to observe that the remaining three subjects of group (a) (Nos. 3a, 5a and 24a) whose mean values (for percentage of cornified cells) did not reach 7% were all of an age at which the onset of the menopause might reasonably/

reasonably be expected to occur, their respective ages being 49 years 2 months, 46 years 10 months, and 51 years.

The results obtained from this investigation of vaginal smears may therefore be said to supply no evidence whatsoever that the performance of hysterectomy on a premenopausal woman will produce a premature cessation of oestrogenic activity on the part of the conserved ovary.

There is moreover, considerable evidence, from the relatively high percentages of cornified cells exhibited in the smears of many of the subjects of group (a), to indicate that the degree of oestrogenic activity manifest by a woman who has undergone hysterectomy with conservation of one or both ovaries, may, two, three, or even six years later, be indistinguishable from that of a normal woman of the same age group, who has undergone no pelvic operation.

## (2) Bio-assay of the Pituitary Gonadotrophin

Before considering the results that were obtained from the estimation of the pituitary gonadotrophin of the urine of those subjects whose ovaries were conserved at hysterectomy, it is necessary to emphasise the fact that an interval of time varying from under one year in some cases to well over two/

two years in others, intervened between the performance of this investigation and the one that has just been discussed.

Some of the subjects who were investigated by both methods had at the time of the former test, reached an age when they were considered to be probably on the threshold of the menopause. It is not surprising, therefore, that the results returned in this later investigation, by certain subjects, should suggest that there had occurred in the interim, a decline in the oestrogenic activity of the ovaries, which is reflected in significantly high values for the pituitary gonadotrophin of the urines of these individuals.

Of the twenty-four subjects of group (a), four (Nos. 17a, 22a, 23a and 24a) as has already been stated, did not submit samples of urine for pituitary gonadotrophin bio-assay. The oestrogenic activity of their ovaries must, therefore, be judged on the results of the fourth investigation (i.e. that concerned with vaginal smears) without the confirmatory evidence of the fifth and final test.

It was noted prior to the performance of this last investigation that of the twenty remaining subjects, five reported that within the past year they had experienced vasomotor/



vasomotor attacks. As all these women had passed the average age of onset for the menopause in a British subject, the information was received without surprise, except for the fact that one of them (No. 21a) had previously complained of this symptom in the few weeks immediately following hysterectomy. The more recent attacks, she insisted, were however, much more severe in character. This phenomenon will receive special comment in a later part of this discussion. The remaining four women had not hitherto experienced this symptom.

The values for pituitary gonadotrophin obtained from the urines of these five women range from 201 to 910 units HMG.20A with a mean value for this subgroup of 433.2 units.

On the other hand, the values obtained from the urines of the remaining fifteen subjects of group (a), all of whom were under the age of 48 years, ranged from 23 to 63 units HMG.20A with a mean value for the subgroup of 41.26 units.

When it is recalled that the range of values obtained from the hysterectomised and castrated subjects of group (b) ranged from 118 to 552 units HMG.20A with a mean group value of 301.9 units, the significantly lower order of the values returned by those women of group (a) who had not yet reached the/

the age of 48 years, is at once obvious.

The relatively lower values which have been consistently returned by this subgroup, strongly suggest that some common factor differentiates each of these fifteen women both from the five older members of group (a) and also from the subjects of group (b).

By far the most probable explanation, and one that is consistent with the accepted views on the interaction of oestrogen and pituitary gonadotrophin is that the ovaries of each of these fifteen women were still functioning when this test was undertaken, and that their output of oestrogen was sufficiently great to ensure that the gonadotrophin of the pituitary was maintained at relatively low values.

The results of this investigation in effect provide strong evidence that the oestrogen production of the ovaries of those fifteen subjects of group (a) was still at pre-menopausal level, and that the operation of hysterectomy which each of these women had undergone at times varying from 2 years 7 months to 7 years 10 months earlier, had not abolished this form of ovarian activity.

It is noteworthy that when the vaginal smears of these fifteen/

fifteen subjects were investigated, the values returned for the mean percentage of cornified cells found in a series of 28 smears was, in every case, within the range of values returned by the normal premenopausal subjects of group (c).

Of the five subjects of group (a) whose pituitary gonadotrophin values confirmed the impression that they had probably reached the menopause, three (Nos. 4a, 6a and 21a) had previously been classified as a result of the investigation of their vaginal smears, as exhibiting evidence of oestrogenic activity comparable to that of the normal subjects of group (c).

It is necessary to add, however, that the intervals between the two investigations were respectively 2 years 3 months, 1 year 10 months and 1 year 3 months, so that the results cannot be considered as necessarily contradicting each other.

The most probable explanation would appear to be that in the intervals, each of these subjects each entered upon a menopause which in no case could be said to be premature, and which could not logically be related to the hysterectomy which she had undergone some years earlier.

The values returned for the mean percentage of cornified cells, by the two remaining women of this subgroup (Nos. 3a and 5a)/

were both observed to be well below the average figure for the normal subjects of group (c), so that even when their vaginal smears were being investigated, both subjects showed evidence of a decline in the oestrogen producing powers of their ovaries.

It is scarcely surprising therefore, that when intervals of 16 and 27 months respectively had elapsed, they should be found to return values for pituitary gonadotrophin of the urine which strongly suggested a further decline in oestrogenic activity - a decline sufficiently pronounced to justify the assumption that both women had in the interim reached the menopause.

It may therefore be said that the results of these two investigations offer no evidence that the performance of hysterectomy with conservation of one or both ovaries will produce a premature decline in the oestrogen producing powers of the ovary.

The evidence taken as a whole would in fact, appear to indicate that women, one or both of whose ovaries have been preserved at hysterectomy (whether it be total or subtotal in character) reach a physiological menopause at about the time/

time when this would be expected to occur, had no operation been performed.

#### Continuation of Ovulation

The second and third investigations were undertaken with the object of discovering whether or not those women who had undergone hysterectomy some time previously still continued to ovulate, and it is on the results of both of these tests considered together, that a conclusion has in each case been reached.

Table XXIV which summarises the evidence presented by the character of the temperature records of the various subjects and the results of the pregnanediol estimations of the urine of these subjects, shows that nine of the women of group (a) were classified as "probably ovulating" at the time when these tests were undertaken, and that in another seven subjects there was reason to believe that ovulation might also be occurring, although the results in the latter group were less convincing.

Among those subjects in whom the evidence of continuing ovulation was strong, the interval of time that had elapsed since hysterectomy varied from one to six years.

Although/

Although neither the fourth nor fifth investigations which followed the ones just mentioned, were capable of giving specific indication of the presence or absence of ovulation, it is significant that, of the nine subjects considered to be ovulating on the basis of the second and third investigations, eight were later found to return relatively low values for pituitary gonadotrophin. These later results may be said to be consistent with the earlier ones, and it is not impossible that, at the time when the fifth investigation was undertaken, ovulation may still have been occurring in some or all of these eight subjects. That subject who forms the exception (No. 21a) had, as has already been suggested, probably reached the menopause by the time the final test was conducted. It is interesting however to observe that she was already 48 years of age when, by temperature record and pregnanediol estimation, she was still considered to be ovulating.

Of the seven subjects classified as "possibly ovulating", five were subsequently found to produce low values for pituitary gonadotrophin.

One (No. 17a) did not participate in the final investigation/

investigation, and the remaining subject (No. 16a) returned a high value of a degree found in menopausal women, when her urine was estimated for pituitary gonadotrophin eighteen months later.

It may be stated therefore, that the study of the patients' temperature records and quantitative estimation of their urines for pregnanediol, together provide strong evidence that, in women who have undergone the operation of hysterectomy with partial or complete conservation of the ovaries, ovulation may continue for years, and that since the incidence of anovular menstruation is known to become greater among women in the fifth decade (Sharman 1955), there is no evidence on the basis of these results to indicate that the operation of hysterectomy interferes with the capacity of the conserved ovary to ovulate.

#### The Subjective Evidence

While an examination of the results of the first investigation reveals a general agreement with the results of those which followed it, there is good reason to maintain that symptomatology very imperfectly reflects ovarian function.

It/

It is true that the group menopausal index of the castrated subjects (20.42) was found to be significantly higher than that of the group who had undergone hysterectomy with some degree of ovarian conservation (5.92). It should be noted however, that although subjects 2a and 13a were each found to have the relatively high menopausal index of 14, both were considered to have functioning ovaries on the basis of all four of the remaining tests.

To some extent the discrepancy is attributable to the fact that, in the determination of the menopausal index, points are allotted for symptoms that may have no association with the menopause.

Even "hot flushes", regarded by some clinicians as virtually diagnostic of the menopause, may be a faulty indicator of the state of ovarian activity.

The results of subject 13a for example, cast considerable doubt on the reliability of vasomotor symptoms as evidence of ovarian decline, for although this woman stated, with great conviction, that she had suffered from "hot flushes" following the operation of hysterectomy, not only was her temperature record found to be recurrently diphasic, but all other/



other objective evidence was strongly in favour of continued ovarian activity.

Since, as already stated, this subject, on whom a subtotal hysterectomy had been performed some seven years earlier, continued to "menstruate" regularly for about one day each month, there is little, if any reason for believing that her ovaries had ceased to function.

The history of a recurrence of "hot flushes" given by subject 21a suggests that during her convalescence following operation, a woman may experience vasomotor symptoms which she may interpret as "hot flushes". The fact however, that this subject, whose ovarian activity some time after hysterectomy appeared, on the basis of objective evidence, to be far from negligible, experienced a renewed attack of "hot flushes", adds considerably to one's misgivings concerning the diagnostic value of this symptom. Since the second attack was supported by other evidence, in the form of a high figure for pituitary gonadotrophin, it would appear reasonable to assume that the more recent phase of vasomotor symptoms did, in fact, represent the onset of the menopause, whereas her previous experience of vasomotor/

vasomotor instability may have been no more than a transient postoperative phenomenon possibly associated with a state of general debility.

#### A Note on Operative Technique

Several writers have postulated a relationship between the duration of ovarian activity following hysterectomy and the technique employed by the surgeon in performing this operation. Richards (1951) for example, suggests that any procedure which puts tension on the long slender ovarian vessels may bring about thrombosis and thus destroy completely the ovarian blood supply. He therefore recommends that, after the uterus has been removed, the pelvic peritoneum should be closed without tension, so that the ovaries are left as near as possible to their normal anatomical position "in the ovarian fossa on the lateral walls of the pelvis".

Aldridge and Meredith (1950) make a similar case when they state that the incidence of postoperative menopausal symptoms is relatively greater after the subtotal than after the total hysterectomy, a fact which they attribute to the habit most surgeons have of fixing the proximal ends of the broad/

broad and round ligaments to the vaginal vault, in order to facilitate peritonisation, or as a means of support to the vagina, when performing the subtotal operation.

It is perhaps therefore worthy of mention that, at the hospital where all the subjects of these investigations underwent hysterectomy, the practice is that if the ovaries are to be conserved, the stumps of the broad and round ligaments are applied to the vaginal vault.

On the evidence of the results of these investigations the adoption of this point of surgical technique would not appear, per se, inevitably to result in loss of function on the part of ovaries that have been preserved.

It may well be however that, in the past, apparent loss of ovarian activity following hysterectomy has wrongly been attributed by certain authors to removal of the uterus, whereas the true explanation may have been that inadequate care had been taken to ensure that the blood supply to the conserved ovaries had not been impaired.

CONCLUSIONS REACHED ON THE RESULTS OF  
ALL INVESTIGATIONS

The object of these investigations was to obtain information concerning the character, degree and duration of ovarian activity in women who had, prior to the menopause undergone the operation of total or subtotal hysterectomy with partial or complete conservation of the ovaries.

For this purpose, three groups of subject were investigated. These were:-

- (1) group (a) consisting of 24 women who had, some time previously and prior to the menopause, undergone the operation of hysterectomy with conservation of one or both ovaries.
- (2) group (b) consisting of 24 women of comparable age and pelvic pathology, who had prior to the menopause, undergone the operation of hysterectomy with bilateral oophorectomy, and
- (3) group (c), consisting of 14 women, none of whom had yet reached the menopause, or had undergone either hysterectomy or oophorectomy.

The investigations were five in number and consisted of:-

- (a) the determination of the menopausal index of each subject and each group of subjects.
- (b) the examination and classification of the basal temperature records of each subject, in order to ascertain whether or not she exhibited the monophasic or the diphasic pattern, the latter if definite and recurrent constituting evidence of ovulation on the part of that particular subject.

- (c) the estimation of the pregnanediol content of 48-hour samples of urine, collected at times selected in accordance with the pattern of the temperature record of each subject and used as a method of re-assessing the accuracy of the conclusions reached by the previous method.
- (d) the collection and microscopical examination of a series of vaginal smears from subjects of all groups, with the object of determining the degree of oestrogenic activity (if any) manifest in these smears.
- (e) the determination, by bio-assay, of the pituitary gonadotrophin of the urine of (1) 20 subjects who had undergone hysterectomy with conservation of one or both ovaries, and also of (2) 20 subjects who had undergone hysterectomy with bilateral oophorectomy.

From the results of these investigations, certain conclusions emerge.

(1) Evidence obtained by vaginal smear and gonadotrophin estimation demonstrates that the oestrogenic activity of ovaries conserved at hysterectomy persists for years. There is, moreover, no indication that hysterectomy hastens the onset of the menopause. Subjects showing evidence of decline in the oestrogenic powers of the ovary were found to be of an age when the onset of the menopause was to be expected. An attempt to assess oestrogenic activity quantitatively by means of vaginal smear, showed that the degree of oestrogenic potency manifest in the vaginal smears of/

of women who had undergone hysterectomy with partial or complete ovarian conservation, was comparable to that of normal women of the same age group, and that this degree of oestrogenic activity might be found in hysterectomised subjects one, two, or even six years after the uterus had been removed.

(2) Evidence obtained by means of basal temperature record and pregnanediol estimation of the subjects' urine, indicated that 9 of the 24 subjects who had undergone hysterectomy with some degree of ovarian conservation might justifiably be assumed to be still ovulating at the time when these investigations were undertaken, i.e. up to seven years after hysterectomy.

Another 7 subjects of the same group also gave indication of continued ovulation, though the evidence here was somewhat less convincing.

Seven women of this group showed no evidence of continued ovulation, and the results of one subject were inconclusive.

As most of the subjects of this, the main investigation group, were over 40 years of age, and as anovular cycles tend to become more frequent as a woman approaches the menopause, it/

it is not unreasonable to suppose that the incidence of ovulation among those women whose ovaries had been preserved at hysterectomy did not differ significantly from that obtaining in normal women.

(3) Although symptoms usually associated with the menopause were found to be more prevalent among the castrated subjects than among those who had undergone hysterectomy with conservation of one or both ovaries, menopausal symptoms were found to be an unreliable indicator of the state of the ovarian activity of the individual.

(4) It is suggested that the apparent loss of ovarian activity which some writers have reported as following hysterectomy, may have arisen from failure to ensure that the conserved ovary is left with an adequate blood supply. Ovarian atrophy and loss of function is not however, likely to be a necessary consequence of applying the stumps of the round and broad ligaments to the vaginal vault, since this practice is followed at the hospital in which the subjects of these investigations underwent hysterectomy, and, as the foregoing pages of this thesis indicate, continued ovarian activity is manifest in the majority of these subjects years after operation.

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**P A R T    I I**

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PATHOLOGY and the CONSERVED OVARYA STATISTICAL SURVEYI N T R O D U C T I O N

In the general introduction to this thesis, it was stated that certain gynaecologists invariably remove the ovaries when performing the operation of hysterectomy. Their two main arguments for so doing have been mentioned, and the purpose of the preceding tests has been to examine, in some detail, the validity of one of them.

It is perhaps fitting, therefore, that before these investigations are concluded, some brief consideration be given to the second argument, which is that when hysterectomy is performed the ovaries should be removed, so that the patient may not, in the future, suffer from any pathological condition of these organs, and, in particular, from ovarian malignant disease.

It would be useful to obtain a precise statistical assessment of the possibility of pathological change, and in particular, of malignant disease in the conserved ovary, but this information is unfortunately not available, and such literature from the English-speaking world as throws any light on this subject would appear to be predominantly/

predominantly, if not exclusively, American.

Recent Literature on the Subject

J.L. Reycraft (1955), in a survey of the statistical records of the Cleveland University Hospitals from the years 1944 to 1953, found only nine cases of carcinoma arising in ovaries which had been conserved when the uterus had been removed, an estimated incidence of .2%.

Counsellor Hunt and Haigler (1955), approaching the question from another aspect, have surveyed "more than 1500 "cases of proved carcinoma of the ovary" occurring between the years 1930 and 1952, and have found that sixty-seven of the patients, or 4.5% had already undergone the operation of hysterectomy for a benign condition. Though less reassuring than Reycraft's figures, to those whose habit it is to preserve the apparently healthy ovary, these statistics give no real impression of the incidence of malignant disease arising in ovaries that have been conserved at hysterectomy, since they give no idea of the number of hysterectomies with conservation of ovaries which were performed to produce these sixty-seven cases of ovarian carcinoma.

Rendall and Gerhardt (1954), basing their assessment on the statistics of the Health Departments of the States of Connecticut and New York (excluding New York City), have calculated that the probability of a woman who has attained the age of forty developing carcinoma of the ovary is .9%.

A search for comparable British statistics on this subject has proved to be quite fruitless. It was therefore decided to conduct a survey of this problem based on the records of a large British teaching hospital.

#### METHOD

The records of one of the units of the Glasgow Royal Samaritan Hospital for the years 1927 to 1948 were examined, as well as the records of all three units for the years 1948 to 1955. From these examinations two distinct surveys have been made.

In the first, a comprehensive list has been compiled of every case in which a hysterectomy has been performed with conservation of one or both ovaries, and the records of each of these patients have been carefully and individually scrutinised/

scrutinised to determine whether or not the patient was subsequently readmitted and found to be suffering from disease of the ovaries. Where this was observed to have occurred, the date and circumstances of the occasion were noted.

In the second survey, a list was made of all cases of oophorectomy and ovariectomy, as well as of laparotomy for what appeared to be ovarian pathology. Once again the case records of each woman were individually examined to ascertain if the patient had previously undergone the operation of hysterectomy, and where this was so the details were recorded.

The work was facilitated by the fact that, in this particular hospital, the patients' clinical notes are entered in a case record in which, under a printed heading, space is specifically allotted to previous operations (nature, place and date). As this form of case record has, for thirty years, been in use in the hospital, and as successive house surgeons have been obliged to complete these case records, the possibility of a previous hysterectomy not being mentioned in the notes becomes very slight. Moreover, in the Glasgow Royal Samaritan Hospital, a patient admitted for/

for the first time is given a number in the clinical records of the hospital, and should she be readmitted at a subsequent date, the notes relating to her readmission are filed under the same number. All records dealing with the various occasions on which she may have been admitted to this hospital are therefore filed in one folder under one number. Admissions, previous and subsequent to a specific date, are therefore easy to obtain.

As the card index system had been in operation in one of the units of this hospital since the year 1927, permission to examine the records of that particular unit was requested. This request was kindly granted.

The records of the other two units were not scrutinised over an equally long period, as in neither had the card index system been in operation until comparatively recently.

From 1948 onwards however, the very comprehensive card index system of the Pathology Department made it easy for one to obtain such information as was required about all cases of hysterectomy and oophorectomy from all three units of the hospital.

R E S U L T S

In the former of these two investigations, the records of one of the three units of the Glasgow Royal Samaritan Hospital for Women for the years 1927 to 1955 were surveyed, as well as the records of the two remaining units for the years 1948 to 1955 inclusive. These two sources together produced a total of 1,215 cases in which hysterectomy had been performed with conservation of one or both ovaries.

In only four of these cases had the patient been readmitted to hospital at a later date on account of ovarian pathology (Nos. 216, 300, 338 and 469) and there was no instance of a readmission due to a conserved ovary having undergone malignant change.

A parallel investigation, covering the same years and units was also conducted, in which the records relating to all cases of oophorectomy and ovariectomy were examined, to discover whether or not the patients had previously undergone the operation of hysterectomy. Not only so, but in order that no case of inoperable carcinoma of the ovary might be excluded, all records of laparotomies were read carefully to ascertain/

ascertain whether or not the patient had, at some previous date, had her uterus removed.

Records of cases in which uterus and ovaries were concurrently excised were not examined, since in these the possibility of the patient having had a previous hysterectomy did not exist.

The total number of cases thus disclosed was 872, and in four instances the records revealed that the patient had previously undergone the operation of hysterectomy (Nos. 229, 384, 530, 774). One of these (No. 774) was a case of adenocarcinoma (of unknown origin) occurring in the right ovary of a woman whose left ovary and uterus had previously been removed. In two other cases the ovarian condition was benign (Nos. 384 and 530), and in the remaining one (No. 229), there was no pathological report on the excised ovary, but the operation notes give no hint that malignant disease was even suspected.

Table XXXIII

Cases of Hysterectomy with Conservation of One or Both Ovaries	
Total Number of Cases	1,215
Number of Readmissions for Ovarian Pathology	4 (.33%)
Number of Readmissions for Malignant Disease of the Ovary	Nil

Table XXXIV

Cases of Oophorectomy and Ovariectomy plus Cases of Laparotomy with Possible Ovarian Pathology	
Total Number of Cases	872
Number having had Previous Hysterectomy	4 (.46%)
Number of Cases of Malignant Ovarian Disease having had Previous Hysterectomy	1

### D I S C U S S I O N

It cannot be claimed that the former of these two surveys proves conclusively that of 1,215 women who had hysterectomies performed with conservation of ovaries, not a single one has subsequently developed malignant disease of the ovary. That one or more of these women may have been admitted, for such a condition, to another hospital in this or another country, is, of course, possible. The figures are nevertheless impressive, as the Glasgow Royal Samaritan Hospital draws its patients from a very large area in the West of Scotland. It is also safe to assume that, had any of these women developed ovarian disease at a subsequent date, she would, if still resident in the same area, very probably have been readmitted/



readmitted to the Samaritan Hospital.

It is, of course, a valid criticism of this investigation that it cannot attempt to assess the percentage of those 1,215 women who are still alive and may yet develop malignant disease of the ovaries, and it is appropriate to recall that the follow-up becomes progressively shorter as one approaches the year 1955.

When, however, all the limitations of this survey have been considered, it remains a significant fact that careful examination of the records of 1,215 women who had hysterectomies performed during the past 29 years failed to reveal one single instance of a patient having to be readmitted for malignant disease of the ovary and only four who required to have an ovary removed for a benign pathological condition.

In the second survey an attempt was made to gain some idea of the incidence of previous hysterectomy among women suffering from ovarian disease which required surgical treatment.

That group of cases in which uterus and ovaries were together removed because of ovarian pathology was not determined, the survey being restricted to cases of oophorectomy/

oophorectomy and ovariectomy, as well as to a few cases where laparotomy alone was performed and in which the pathology was possibly ovarian.

Of the 872 women included in these categories, it was discovered that three who suffered from innocent ovarian conditions had previously had the uterus removed, a disclosure which can scarcely be said to justify the routine excision of ovaries when hysterectomy is performed.

While it is true that the records also revealed one case of adenocarcinoma of the ovary in a patient who had previously had her uterus and other ovary removed, it must also be noted that the pathologist recorded his inability to identify the origin of the tumour, which may therefore have been secondary to a malignant growth in another organ.

#### SUMMARY and CONCLUSIONS

The object of this survey was to obtain statistical information concerning the incidence of pathological change, and in particular of malignant change, in ovaries conserved at hysterectomy.

The/

The subject is an important one as the possibility of the conserved ovary subsequently becoming diseased has been used to justify routine oophorectomy when the uterus has to be removed for a benign condition.

Precise information on this question is difficult to obtain, and no important investigation of this problem would appear to have been undertaken recently in this country.

Reycraft (1955) in a survey of the records of the Cleveland University Hospitals from 1944 to 1955, found 9 cases of carcinoma in ovaries which had been conserved at hysterectomy, an incidence of .2%.

Counsellor Hunt and Haigler (1955) surveyed 1,500 cases of proved carcinoma of the ovary, and found that 67 of these occurred in women who had previously had the uterus removed for a benign condition.

Rendall and Gerhardt (1954), basing their calculations on the statistics of the Health Departments of the States of Connecticut and New York (excluding New York City), estimate that the likelihood of a woman who has reached the age of forty developing carcinoma of the ovaries is .9%.

In a survey of 1,215 cases of hysterectomy with conservation/

conservation of one or both ovaries performed during the years 1927-1955 in the Glasgow Royal Samaritan Hospital for Women, it was found that four of the patients were subsequently readmitted to that hospital on account of ovarian pathology. No case of readmission for malignant ovarian disease was discovered.

In a parallel series of 872 cases of oophorectomy, ovariectomy, and laparotomy for inoperable conditions which may have been ovarian, it was found that four of the patients had previously had the uterus removed. One of these was a case of adenocarcinoma of the ovary but doubt was expressed by the pathologist concerning the origin of the tumour.

While the limitations of these surveys are appreciated and stated, it is felt that they indicate that the risk of malignant disease developing in ovaries conserved at hysterectomy has probably been exaggerated by some gynaecologists who argue that when hysterectomy is performed for a benign condition, the ovaries ought also to be removed lest they should subsequently undergo neoplastic change.

BIBLIOGRAPHY

## A

- (1) Aldridge A. H. and Meredith R. S. (1950)  
Amer. Jour. Obst. and Gyn. 59,748.
- (2) De Allende I. C. and Oriás O. (1950A)  
Cytology of the Human Vagina. Paul B. Hoeber  
Inc. New York. Pages 19-20.
- (3) De Allende I. C. and Oriás O. (1950B)  
Cytology of the Human Vagina. Page 26.
- (4) De Allende I. C. and Oriás O. (1950C)  
Cytology of the Human Vagina. Pages 149-157.
- (5) De Allende I. C. and Oriás O. (1950D)  
Cytology of the Human Vagina. Page 51.

## B

- (1) Bancroft-Livingston George (1954)  
Jour. Obst. and Gyn. Brit. Emp. Vol.61 p.628-638.
- (2) Barton M. and Wiesner B. P. (1945A)  
Lancet 2: p. 663-669.
- (3) Barton M. and Wiesner B. P. (1945B)  
Lancet 2: p. 671-672.
- (4) Beall D. (1939)  
Nature 144, 76.
- (5) Bishop P. M. F. (1938)  
Brit. Med. Jour. 1: 939-941.
- (6) Bishop P. M. F. (1947)  
Gynaecological Endocrinology for the Practitioner  
1st Edition. Pages 51-54. E. and S. Livingstone,  
Edinburgh,

- (7) Bishop P. M. F. (1950)  
Modern Trends in Obst. and Gyn. P.586-607.  
Butterworth and Co. London.
- (8) Block K. (1945)  
J. Biol. Chem: 157. 661-666
- (9) Bonine R. G. (1950)  
Amer. Jour. Obst. and Gyn. Vol. 60.  
p. 1306-1314.
- (10) Bonney Victor (1937)  
Jour. Obst. and Gyn. Brit. Emp. Vol. 44 p.1-12.
- (11) Browne, J. S. L., Henry, J. S. and Venning E.M. (1937)  
Journ. Clin. Investigation. Vol. 16. p. 678.
- (12) Burford T. H. and Diddle A. W. (1936)  
Surgery, Gynaecology and Obstetrics. Vol. 62.  
p.701-707.
- (13) Butenandt A. (1930) Berichte der Deutschen  
Chemischken Gesellschaft Vol. 1 p. 659-663.
- (14) Buxton C. L. and Atkinson W. B. (1948)  
Jour. Clin. Endo. 8: 544-549.
- (15) Buxton C. L. and Engle E. T. (1950).  
Amer. Jour. Obst. and Gyn. Vol. 60: 539-551.
- (16) Buxton C. L. and Westphal U. (1939)  
Proc. Soc. Exper. Biol and Med. 41: 284-287.

## C

- (1) Callow R. K. and Emmens C. W. (1940)  
Jour. Endocrin. 2: 88.
- (2) Council of the Medical Women's Federation (1933)  
Lancet 1: p. 56.
- (3) Counsellor V. S., Hunt, W, and Haigler F. H. (1955)  
Amer. Jour. Obst. and Gyn. Vol. 69, p. 538-542.

- (4) Cummings Howard H. (1954)  
Obst. and Gyn. Survey Vol. 9 p. 51-52.
- (5) Cuyler W. K., Ashley, C. and Hamblen E. C.  
Endocrinology Vol. 27, p. 177-178.

## D

- (1) Davis M. E. and Fugo N. W. (1948)  
Jour. Clin. Endo. Vol. 8, p.550-563.
- (2) Dodds E. R. and Noble R. L. (1936)  
Brit. Med. Jour. 2: 824-826.

## E

- (1) Elert R. (1951)  
Geburtshilfe und Frauenheilkunde 11: 325-328.
- (2) Engelhart E. (1930)  
Klin. Wchnschr. 9: 2114.
- (3) Englehart E. (1932)  
Arch. für Gynäk 149: 688.
- (4) Englehart E. (1935)  
Klin. Wchnschr. 14: 1068.

## F

- (1) Fevold H. L., Hisaw, F. L. and Leonard S. L. (1931)  
American Jour. Physiol. Vol. 97: p. 291-301.
- (2) Fraenkel L. (1903)  
Arch. für Gynäk. Vol. 68: p. 438-545.
- (3) Fraenkel L. and Papanicolaou G. N. (1938)  
Amer. Jour. Anat. Vol. 62: p. 427-451.
- (4) Frank R. T., Salmon U. J. and Friedman R. (1935)  
Proc. Soc. Exp. Biol and Med. Vol. 32, p.1666-1667.

## G

- (1) Giles A. E. (1897)  
Transactions of the Obstetrical Society of  
London, Vol. 39, p. 115-124.
- (2) Greenblatt R. B., Barfield W. E., Garner J. F.,  
Calk. G. L. and Harrod J. P. (1950)  
Jour. Clin. Endo. Vol. X, p. 1547-1558.
- (3) Greulich, W. W., Morris E. S. and Black M. E. (1943)  
Proc. Conf. on Problems of Human Fertility  
p. 37, Banta Publishing Co. Wisconsin.

## H

- (1) Hadfield G. (1956)  
Brit. Med. Jour. p. 1507-1511, 1956.
- (2) Halbrecht I. (1945)  
Lancet 2: 668-669, 1945.
- (3) Halbrecht I. (1947)  
Jour. Obst. and Gynaec. Brit. Emp. Vol. 54: 848-852.
- (4) Hamblen E. C., Ashley C, and Baptist M. (1939)  
Endocrinology Vol. 24: p.1-12.
- (5) Hawkinson L. F. (1938)  
Jour. Amer. Med. Assoc. Vol. III, p.390-393.
- (6) Heller C. G., Farney J. P. and Myers G. B. (1944)  
J. Clin. Endo. Vol. 4: p.101-108.
- (7) Hendry J. (1936)  
Jour. Obst. and Gynaec. Brit. Emp. Vol. 43, p.609-618.
- (8) Hobson B. M. (1952)  
Quarterly Jour. Exper. Physiol. Vol. 37, p.191-203.
- (9) Hobson B. M. and Landgrebe F. W. (1954)  
Quarterly Jour. Exper. Physiol. Vol. 39, p. 23-27.



## I

- (1) Israel S. and Schneller O. (1948)  
Urol. and Cutan. Rev. 52: 630-632.

## J

- (1) Jones G. E. S. and Te Linde R. W. (1941)  
Amer. Jour. of Obst. and Gyn. Vol. 41,  
p.682-687.

## K

- (1) Katzman P. A. and Doisy E. A. (1934)  
Jour. Biological Chem. Vol. 106, p.125-139.
- (2) Kernodle J. R. and Cuyler W. K. (1948)  
Southern Medical Journal Vol. 41. No. 10,  
p. 869-872.
- (3) Kemp, T. and Pedersen-Bjergaard K. (1937)  
Lancet 233: 842.
- (4) Kleitman N. and Doktorsky A. (1933)  
Amer. Jour. Physiol. 104: 340.
- (5) Kleitman N. Cooperman N. R. and Mullin F. J.  
Amer. Jour. Physiol. 105: 574.
- (6) Klopper A., Michie E. A. and Brown J. B. (1955)  
Jour. Endocrin. Vol. 12, p.209-219.
- (7) Klopper A. (1956)  
Personal Communication.
- (8) Krane W. (1937)  
Archiv. für Gynäk 164: 101-132, (Archiv für  
Gynäkologie).
- (9) Kretschmar N. R. and Gardiner S. (1935)  
Amer. Jour. Obstet. and Gyn. Vol. 29, p.168-175.

- (10) Kupperman H. S. Meyer H. G. B., Wiesbader H. and Fuller W. (1953)  
 Jour. Clin. Endo. Vol. 13(1) p. 688-703.

## L

- (1) Langreder W. and Zimmerer G. (1953)  
 Arch. Gynák. Vol. 184, p.1-13.
- (2) Te Linde R. W. (1942)  
 Jour. Amer. Med. Association Vol. 118, No. 16  
 p.1341-1345.
- (3) Loraine J. A. (1952)  
 Jour. Obst. and Gyn. Brit. Emp. Vol. LIX No. 4  
 p.535-557.
- (4) Loeb L., Saxton J. and Hayward S. J. (1936)  
 Endo. Vol. 20. p.511-519.

## M

- (1) Magallon D. T. and Masters W. H. (1950)  
 Jour. Clin. Endo. Vol. X, p.511-518.
- (2) Marrian G. F. (1929)  
 Biochem. Jour. Vol. 23 (2) p.1090-1098.
- (3) Martin P. L. (1943)  
 Amer. Jour. Obst. and Gynec. Vol. 46, p.53-62.
- (4) Marx R., Catchpole H. R. and McKennon B. J.  
 Surg. Gyn. Obst. Vol. 63: 170-177, 1936.
- (5) McLaren H. C. (1941)  
 Jour. Obst. and Gyn. Brit. Emp. Vol. 48, p.1-40.
- (6) Medical Women's Federation (Council of) (1933)  
 Lancet Vol. 1, p.106-108.
- (7) Murray M. and Osmond-Clarke F. (1956)  
 Brit. Med. Jour. p.157, July 21, 1956.

## N

- (1) Nieburgs H. E. (1946)  
Lancet Vol. 1, 627-628.
- (2) Nieburgs H. E. and Greenblatt R. B.  
Jour. Clin. Endo. Vol. 8, p.622.
- (3) Novak, E. (1939)  
Amer. J. Obst. and Gyn. Vol. 37, p.605-616.
- (4) Novak E. (1941)  
Gynaecological and Obstetrical Pathology  
p.2 W.B. Saunders Company, Philadelphia & London.
- (5) Noyes R. W., Hertig A. T. and Rock J. (1950)  
Fertil and Steril Vol. 1 p.3-25 (Jan. 1950).

## P

- (1) Palmer A. (1950)  
Amer. J. Obst. and Gyn. Vol. 59, p.155-161.
- (2) Palmer R. and Devillers J. (1939)  
Compt. rend. Soc. de biol. Vol. 130, p.895-896.
- (3) Perlman R. M. (1948)  
J. Clin. Endo. Vol. 8, p.586.
- (4) Papanicolaou G. N. (1933)  
Amer. Jour. of Anatomy Vol. 52, No. 3 Supplement  
p.519-616. May 1933.
- (5) Papanicolaou G. N. and Shorr E. (1936)  
Amer. Jour. Obst. and Gyn. Vol. 31, p.806-831.

## R

- (1) Randall C. L. and Gerhardt P. R. (1954)  
Amer. Jour. Obst. and Gynec. Vol. 68, p.1378-1387

- (2) Reycraft J. L. (1955)  
Amer. Jour. Obst. & Gynec. Vol. 69, p.543-544.
- (3) Reynolds S. R. M.  
Physiology of the Uterus, Chapter 35, p.503-510  
2nd Edition. Paul B. Hoeber, New York.
- (4) Richards N. (1951)  
Proceedings Royal Soc. Med. Vol. 44, p.496-498.
- (5) Rogers J. and Sturgis S. H. (1950)  
Jour. Clin. Endo. Vol. X, p.89-100.
- (6) Rothchild I. and Allan C. B. (1952)  
Endocrinology Vol. 50, p.485.
- (7) Rubenstein B. B. (1937)  
Amer. Jour. Physiol. Vol. 119, p.635-641.

## S

- (1) Salmon U. J. and Frank R. T. (1936)  
Proc. Soc. Exp. Biol. and Med. Vol. 34,  
p.463-466
- (2) Salmon U. J., Geist S. H. and Salmon A. A. (1941)  
Proc. Soc. Exper. Biol. and Med. Vol. 47. p.279-280.
- (3) Scott L. D. (1940)  
Brit. Jour. Exper. Path. No. 21, p.320-324.
- (4) Sessums J. V. and Murphy D. P. (1932)  
Surgery, Gynaecology and Obstetrics Vol. 55,  
p.286-289.
- (5) Sharman A. (1955) Modern Trends in Obstetrics  
and Gynaecology (Second Series) p.97-100.  
Butterworth & Co. London.
- (6) Shaw W. (1943)  
Textbook of Gynaecology, 3rd Edition, p.92.  
J. & A. Churchill Ltd., London.

- (7) Siegler S. L. and Siegler A. M. (1951)  
Fertility & Sterility Vol. 2 p.287-301.
- (8) Squire W. (1868)  
Transactions of the Obstetrical Society  
of London. Vol. 9, p.129.

## T

- (1) Tamis A. B. (1934)  
Amer. Jour. Obst. and Gyn. Vol. 28, p.48-60.

## V

- (1) Venning E. H. and Browne J. S. L. (1937)  
Endocrinology Vol. 21, p.711-720.
- (2) Venning E. H. and Browne (1938)  
Amer. Jour. Physiology Vo. 123, p.209-210.
- (3) Venning E. H. and Browne J. S. L. (1940)  
Endocrinology Vol. 27 p.707-720.
- (4) Vincent Memorial Hospital (1950)  
The Cytologic Diagnosis of Cancer, p.1-16.  
W.B. Saunders Co. Philadelphia & London.
- (5) Vollmann Ursula Von  
Monatsschrift fur Geburtshilfe und Gynäkologie  
Vol. III, p.121-154.

## W

- (1) Wachtel G. E. (1955)  
Personal Communication
- (2) Wallen-Lawrence Z. (1934)  
Jour. Pharmacology and Exper. Therapeutics  
Vol. 51, p.263-286.

- (3) De Watteville H. Borth R. and Gsell M. (1948)  
Jour. Clin. Endo. Vol. 8, p.982-992.
- (4) De Watteville H. (1951)  
Jour. Clin. Endocrinology Vol. II, p.251-266.

## Z

- (1) Zondek B. (1926),  
Zeitschilfe fur Geburtshulfe Vol. 90, p.372-380.
- (2) Zondek B. (1930)  
Klin. Wchnschr. 9, 964.
- (3) Zondek B. (1932)  
Amer. Jour. Obst. Gynec. Vol. 24, p.836-843.