AN INVESTIGATION OF THE SALT AND WATER BALANCE IN MIGRAINE

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The physiological changes which are believed to occur in migraine were first summarized by Kinnier Wilson (1940) as follows: (1) A vascular disturbance. (2) Exudative changes, i.e., oedema either due to (a) arterial spasm followed by anoxaemia and increased capillary permeability in localized areas of the brain, or (b) of an angioneurotic type set up by an allergic stimulus. (3) A disturbance of the neurosympathetic system. (4) The action of endocrine factors such as pituitary hormones.

In a more recent review of the migraine syndrome Wolff (1948) has shown how the evidence points to a primary constriction of the cerebral vessels, which gives rise to the prodromal signs of, scotomata, vertigo, paraesthesia, and mobility defects; followed by vasodilatation during the period of headache. He quotes an interesting case described by Goltman (1935-6) of a young woman who had a left frontal bone defect following a decompression and who suffered from migraine. Before the onset of headache there was a blanching of the face followed by depression of the tissues within the area of the defect. As the headache developed the cerebral tissues were observed to expand so much as to cause a non-pulsatile swelling through the aperture in the skull. The patient then vomited and the swelling slowly subsided. These changes were visible evidence of an alteration in intracranial volume from one phase of the migraine attack to the next.

As there are no records in the literature of any changes in the cerebrospinal fluid pressure which might account for the cerebral swelling, it is reasonable to suppose that it is part of the generalized oedema which is known to occur before and during the early part of an attack of migraine. Amongst our own cases, which now number over 200, we have observed oedema of various parts of the body before an attack. Women patients often volunteer the information that their skirts become tight or their legs swell; while a number of subjects notice an increase in body weight. One made the remarkable complaint that her dentures fell out before an attack owing to the swelling of her gums.

It is well known clinically, too, that diuresis may occur in the terminal phase of an attack of migraine, but no explanation has been given of this, of the oedema, or of the subsequent restoration of a normal state of hydration.

Our interest in the salt and water balance in migraine arose from the trial by one of us (D.A.C.) of the effects of urea in the treatment of migraine as recommended by Brown (1943). In a series of 66 cases treated with sustained doses of $7\frac{1}{2}$ g. of urea twice daily over a period of at least two months, a definite alleviation of symptoms was reported in 51 cases, which was a very

much better result than that obtained by other recognized forms of medication. In contrast with the effect of such drugs as ergotamine tartrate, which are commonly employed for the immediate relief of an attack by virtue of their action on smooth muscle, it was found that a perseverance of treatment with urea over a long enough period lessened both the frequency and the severity of the attacks in 45 subjects, and procured complete relief in six of this group of patients.

Since the main action of urea is that of a diuretic, these clinical findings seem to indicate that diuresis, whether of natural occurrence or induced by urea, is a physiological means of restoring the body fluids to their normal balance.

Experimental Procedure

Our investigations into the salt and water metabolism of migrainous subjects were divided into two groups: (1) a study of the effects of a water imbibition test; and (2) an analysis of selected constituents in the blood and in the urine of migrainous subjects during the migraine cycle.

Water Imbibition Test.—This test was chosen as a means of showing the effects of the simplest form of diuretic, and a comparison was made of the behaviour of 8 normal and 26 migrainous subjects after the imbibition of a known amount of water. The migrainous subjects were not suffering from an attack of migraine at the time of these tests. Each individual was admitted overnight and was kept resting. Specimens of urine were kept from 8 p.m. The urine passed for four hours before the imbibition test was used for comparison with that passed for four hours subsequently. On the morning following admission a light breakfast of toast and butter and 6 oz. (170 ml.) of water was given at 7 a.m. Tea and other strong diuretics were forbidden. At 9.30 a.m. 1,500 ml. of water was given and taken in 10

TABLE I.—*Diuresis in Normal Subjects after Imbibition of 1,500 ml. of Water*

Subject No.	Sex	Vol. Urine (ml.)		Chloride (mg.)		Sodium (mg.)	
		Before	After	Before	After	Before	After
1 ..	F.	162	1,729	788	842	561	500
2 ..	M.	115	983	531	723	398	253
3 ..	M.	236	1,548	1,033	1,112	1,098	394
4 ..	M.	165	1,496	996	1,231	704	538
5 ..	F.	119	1,491	460	732	382	312
6 ..	F.	284	1,471	1,107	841	804	444
7 ..	M.	188	1,641	946	872	537	375
Average		181	1,480	839	908	641	431

TABLE II.—*Diuresis in Migrainous Subjects (Group I) after Imbibition of 1,500 ml. of Water*

Subject	Sex	Vol. Urine (ml.)		Chloride (mg.)		Sodium (mg.)	
		Before	After	Before	After	Before	After
1 ..	M.	313	1,772	1,848	1,774	970	995
2 ..	M.	244	1,696	1,090	1,832	647	954
3 ..	F.	205	1,744	437	1,217	311	814
4 ..	F.	197	1,624	1,470	1,951	932	1,296
5 ..	F.	221	1,902	1,174	1,940	720	1,250
6 ..	F.	161	1,739	530	1,450	367	890
7 ..	M.	145	1,770	880	1,576	480	893
8 ..	F.	300	1,592	590	995	310	416
9 ..	M.	155	1,818	673	1,334	541	1,026
10 ..	F.	157	1,839	1,005	1,861	938	1,372
11 ..	M.	156	1,527	632	1,751	823	853
12 ..	M.	137	1,828	786	2,311	527	1,098
13 ..	F.	108	1,351	632	1,216	514	739
14 ..	M.	245	1,886	1,084	2,128	881	1,468
15 ..	M.	113	1,362	700	1,505	690	1,173
16 ..	F.	126	1,338	914	803	523	622
Average		186	1,680	902	1,602	636	966

* Suffered from premenstrual migraine attacks.

FIG. 1.—Rate of urine excretion after 1,500 ml. of water by mouth given at 9.30 a.m. — = Average excretion for 16 cases of migraine. ---- = Average excretion for seven normal control subjects.

the method of Kramer and Gittleman (1924) (Eisenman's modification: Wakeman, Eisenman, and Peters, 1927), and chloride by the modified Volhard-Harvey titration method (Volhard, 1878; Harvey, 1910).

Results

Diuresis.—(a) The average total volume of urine excreted in the resting period (four hours before the test)

FIG. 2.—Range of urine excretion after 1,500 ml. of water by mouth given at 9.30 a.m. Area between continuous lines shows range of excretion for 16 cases of migraine. Shaded area shows range of excretion for seven normal control subjects.

minutes. The urine was collected at frequent intervals during diuresis and each sample was analysed separately.

A marked diuresis occurred in both normal and migrainous subjects, but none of them developed a headache. Ten of the migrainous subjects were unable to drink the total amount of water, and were therefore considered separately (group II). The 16 who were able to drink the 1,500 ml. of water are classified as group I.

Estimations.—The volume, specific gravity and reaction were recorded for each sample of urine. Sodium estimations were performed by

the method of Kramer and Gittleman (1924) (Eisenman's modification: Wakeman, Eisenman, and Peters, 1927), and chloride by the modified Volhard-Harvey titration method (Volhard, 1878; Harvey, 1910).

Total Chloride Excretion.—(a) In the resting period the chloride excretion of the migrainous subject was higher than that of the normal. (b) In the period of diuresis the chloride excretion was raised slightly in the normal subject, and was almost doubled in the migrainous subject (Tables I and II and

Fig. 3). (c) The range of chloride excretion was alike before the test, but dissimilar for four hours afterwards (Fig. 4).

Total Sodium Excretion.—(a) In the resting period the average sodium excretion was the same for normal and for migrainous subjects. (b) In the period of diuresis the sodium excretion was reduced in the normal and markedly increased in the migrainous subject (Tables I and II and Fig. 5). (c) The range for sodium excretion was alike before the test, but quite dissimilar for four hours afterwards (Fig. 6).

These findings were repeated in the 10 migrainous subjects (group II) who were unable to consume the total 1,500 ml. of water (Table III). These subjects had a lower output of urine and of sodium and of chloride in the resting period than those in group I, but the proportional increase in output in diuresis was approximately the same (Table IV).

An analysis of the results in terms of milliequivalents per litre (Table V) shows, as one would expect, that the concentration of sodium and chloride is much reduced in diuresis. It also shows that in diuresis the migrainous subject excretes equivalent amounts of sodium and chloride, but that the reabsorption of sodium and chloride falls considerably below that of the normal subject.

We did not induce an attack of migraine in any of our subjects as did Redisch and Pelzer (1943), who stated that 21 out of 29 subjects suffered from an attack of migraine after the ingestion of 1,500 ml. of water followed by a dry meal after a period of four hours.

Variations in Certain Constituents of Blood and of Urine during the Migraine Cycle

During the past three years it has become increasingly apparent to us that the migrainous subject is invariably in

FIG. 3.—Rate of chloride excretion after 1,500 ml. of water by mouth given at 9.30 a.m. — = Average excretion for 16 cases of migraine. ---- = Average excretion for seven normal control subjects.

FIG. 4.—Range of chloride excretion after 1,500 ml. of water by mouth given at 9.30 a.m. Area between continuous lines shows range of excretion for 16 cases of migraine. Shaded area shows range of excretion for seven normal control subjects.

FIG. 5.—Rate of sodium excretion after 1,500 ml. of water by mouth given at 9.30 a.m. —=Average excretion for 16 cases of migraine. ---=Average excretion for seven normal control subjects.

the blood and of the urine for analysis, in the hope that the variations which occur during the cycle of an attack might reveal further evidence of the nature of the biochemical disturbance in migrainous subjects.

(a) *Level of Blood Sodium.*—The normal range of blood sodium is generally accepted as being 320–350 mg. per 100 ml. We found that several of our subjects whom we submitted to the water imbibition test had a blood sodium level of 340 mg. or above, particularly when they were on the brink of an attack. Further analysis (Table VI) showed that the blood sodium level is high in the prodromal phase of an attack, that it often rises above the upper limits of normal during an attack,

a tense, excitable, or overanxious state before and during an attack, and that during the period of recovery he develops a general flaccidity or exhaustion and a predisposition to sleep, and often exhibits diuresis.

In our series of over 200 cases, two-thirds were women, many of whom suffered from premenstrual migraine and from generalized oedema in the premonitory phase.

We selected certain constituents of

and that it returns to normal in recovery and in the interim phase between attacks. We have repeated these observations on numerous occasions and find that the high level of blood sodium is associated with migrainous headache, whether or not the latter is accompanied by vomiting.

(b) *Estimation of Plasma Proteins.*—In order to estimate the state of hydration of the blood during the migraine cycle, records were made of the albumin and globulin contents of the blood. There is no significant variation as between these proteins, but the total proteins (Table VII) are below the normal value of 6.28 g. % (King, Haslewood, and Delory, 1937)

TABLE III.—*Diuresis in 10 Female Migrainous Subjects (Group II) after Imbibition of 1,000–1,400 ml. of Water*

Quantity of Water (ml.)	Subject No.	Vol. Urine		Chloride		Sodium	
		Before	After	Before	After	Before	After
1,000	1	271	1,497	1,388	2,021	1,069	1,283
	2*	60	849	376	885	263	486
	3*	95	1,272	364	837	226	429
	4*	173	1,270	487	975	277	790
	5*	82	902	405	640	322	444
1,100	6*	175	1,020	733	935	633	766
1,200	7†	208	1,590	872	975	709	824
	8*	46	1,361	156	733	117	415
1,250	9*	313	1,315	1,133	1,395	596	886
1,400	10	117	1,351	645	1,297	512	974
Average 1,115		153	1,243	656	1,069	472	730

* Suffered from premenstrual migraine.

† Post-menopausal migraine.

TABLE IV.—*Average Proportional Increase in Diuresis in Comparison with Resting Period*

	Vol. Urine	Sodium	Chloride
Normal	8.2 times	0.67	1.1
Migrainous Group I	9.0 times	1.52	1.78
Migrainous Group II	8.1 times	1.5	1.63

TABLE V.—*Comparison of Sodium and Chloride Output in mEq per Litre*

	No. of Subjects	Sodium		Chloride	
		Resting	Diuresis	Resting	Diuresis
Normal (after 1,500 ml.)	8	154	14.0	136	17.3
Migraine (after 1,500 ml.)	16	149	25.0	136	26.8
Migraine (after 1,000 ml.)	5	141	25.1	125	25.4

TABLE VI.—*Variation of Blood Sodium in Migraine (mg./100 ml.)*

Subject No.	Prodromal Phase	Migraine Attack	Recovery from Attack	Interim
1				338
2				
3	343			
4	338			
5		346		338
6				333
7	363			
8		353		
9	356			
10	362			
11	346			
12		364		346
13		363		344
14	338	363		
15		363		
16	351	363		
17	341	362		
18		370		331
19		354		
20		353		
21		346		
22		346		
23		353		
24		357		
25	352	357	336	332
26	348	361	338	344
27			342	343
28				348
29				338
30		356		
31		348		
		365		334
		373		332
		373		
		368		
		368		
		368		
		370		
Average	349	359	333	339

In subjects 14, 18, 20, 21, and 31 the successive figures during an attack, and in recovery, were obtained at intervals of 24 hours, on successive days.

FIG. 6.—Range of sodium excretion after 1,500 ml. of water by mouth given at 9.30 a.m. Area between continuous lines shows range of excretion for 16 cases of migraine. Shaded area shows range of excretion for seven normal control subjects.

TABLE VII.—*Variation in Total Plasma Proteins in Migraine (g.%)*

Subject No.	Prodromal Phase	Migraine Attack	Recovery from Attack	Interim
1				6.48
2	5.04			
3	5.67			
4			6.11	
5		6.57	6.41	
6				6.08
7	5.7			
8		5.86		
9	6.15			
10	6.11			
11	6.78			
12				6.41
13		5.79		5.65
14	5.86	5.37		
15		5.22		
16	5.27	5.79		5.87
17	5.83	6.61		
18		6.05		
19		5.27		
20		5.64		
21		5.44		
22		6.16	4.73	
23	5.24	5.06	5.84	5.70
24			5.81	6.21
25	5.10	5.85		6.91
26				6.95
27		5.76		6.91
28		6.52		7.25
29		5.85	6.11	6.82
30		5.80		
31		5.86		
		5.34		
		5.33		
		5.27		
		5.38		
Average	5.70	5.71	5.83	6.44

In Subjects 14, 17, 19, 20, 21, and 31 the successive figures during an attack, and in recovery, were obtained at intervals of 24 hours, on successive days.

both before and during an attack, and they rise again to normal value in the interim. A calculation of the colloid osmotic pressure by Govaerts's (1925) original formula ($5.5 \times \text{albumin g.}\% + 1.4 \times \text{globulin g.}\%$) demonstrates the same phenomenon—namely, the average values are low, being 22.7, 23.6, and 22.8, respectively before, during, and after an attack, and return to a normal level of 26.0 in the interim (normal range, 25–30 cm.Hg). It would appear then that the blood is overhydrated throughout an attack of migraine, and that if the appropriate correction were made for this the level of blood sodium would be still higher.

(c) *Level of Blood Potassium.*—The values for potassium during the migraine cycle were within the normal ranges of 16–20 mg. per 100 ml., but they tended to be

low when the sodium values were high (Fig. 7).

FIG. 7.—Inverse variation in sodium and potassium levels in the serum during the migraine cycle (average of 31 cases).

Discussion

The observations which we have been able to make have shown that most of these people are both physically and mentally overactive. They are liable to tension before an attack, and afterwards complain of feelings of muscular weakness and

fatigue, which often culminate in deep sleep. When they awaken they feel fit and well. The weakness and fatigue contrast markedly with the pre-migraine "motor" tension, and it is during the change from one state to the other that there is an accompanying alteration in certain constituents of the blood.

In a series of cases analysed for symptoms by one of us (K. M. H.) two-thirds were women, many of whom suffered from premenstrual migraine. This is of interest because the sex hormones can produce salt and water retention. Thorn *et al.* (1938) found that, in dogs, sodium chloride and water are retained in significant amounts following the injection of oestradiol. They also found that in 50 women between 23 and 26 years of age 24 gained 1 kg. or more in the premenstrual phase, while two lost weight. The increase in weight was accompanied by swelling of the abdomen. With the onset of menstruation there was an increase of salt excretion and a reduction of body weight. Singh, Singh, and Singh (1947) have precipitated migraine in susceptible subjects by the injection of oestrin, and relieved them with progesterone.

Apart from the more frequent occurrence of migraine in women, and the more severe symptoms on the whole in women as compared with men, there is no essential difference between the sexes in the biochemical findings which we have to report.

Our results show that after the imbibition test with 1,500 ml. of water there are striking differences in the excretion of chloride and of sodium by the migrainous subject as compared with the normal subject, but that in their total excretion of water there is no such difference. In both classes of subject the maximum rate of output of chloride occurred immediately before the peak of diuresis, and diuresis ceased five hours after the test. The normal subjects resumed their resting rate of chloride excretion within two and a half hours of the test, but the migrainous subjects showed a rate of excretion which was still above normal seven hours after the test. The total amount of chloride excreted by migrainous subjects during the four-hour test period was about double that excreted by normal subjects, although in the four-hour test period preceding the test the excretion of chloride was much the same as in the normal. Our normal subjects showed a transitory increase and then a decided fall in chloride excretion during diuresis, the variation being the same as that recorded by Marshall (1920), Priestley (1921), and Eggleton (1943), if allowance is made for the different methods employed for testing.

We found an even more striking difference in the sodium excretion. Whereas in the resting period before the test the excretion of sodium was almost precisely the same in the two classes of subjects, during diuresis the normal subjects showed a decrease of approximately one-third in comparison with the resting output, while the migrainous subjects increased their output by one-half.

Relation of Blood Sodium Level to Sodium Excretion

Many of our migrainous subjects showed a relatively high level of blood sodium at the time of the imbibition test, but still exhibited a normal water diuresis. In fact, neither the initial level of the blood sodium nor the extent of its fall during a known period bore any close relation to the amount of sodium excreted or to the total diuresis during the same period. Crutchfield and Wood

(1948) obtained a normal diuresis after giving 1,000 ml. to patients with congestive heart failure, many of whom had a low blood sodium. They concluded that the "retention of the sodium ion is of nearer primary importance than the retention of water." McCance and Widdowson (1937-8), on the other hand, were unable to produce a water diuresis in Addison's disease. These observations, together with our findings that normal water diuresis can occur in the presence of a high blood sodium, support the view that the level of the blood sodium is not the only factor in the production of diuresis. More recently Verney (1946) has shown that water excretion is controlled by the antidiuretic hormone which is produced by the posterior lobe of the pituitary body. He states that the diuresis which follows water imbibition is due to the rapid dilution of the blood, which inhibits the activity of the hormone. This hormone is the primary factor in maintaining the fluid volume of the body at an almost constant percentage of the body weight.

Newburgh and Leaf (1950) point out that the fixity of concentration of sodium and chlorides in the extracellular fluids is a function of adrenocortical hormones working in conjunction with the antidiuretic hormone on the kidneys. "These hormones working together cause a reabsorption of a fluid in which the concentration of sodium is very nearly identical with its concentration in the extracellular fluid, and also the reabsorption of the amount of this fluid which is required to maintain the normal quantity of body fluid." The action of the kidneys in conserving sodium is one which favours the shift of water from intracellular to extracellular compartments of the body and the maintenance of extracellular hydration.

The observations recorded in the second part of our experimental work provide evidence that the state of hydration of the blood and the level of blood sodium are disturbed during the various phases of the migraine cycle. Not only did a number of patients develop oedema as a physical sign of an impending attack of migraine, but they gave evidence of increased hydration of the blood by a fall of total proteins of the blood before, during, and after an attack. The average for each phase was 5.70, 5.71, and 5.83 g.%, respectively. In the interim phase, between the attacks, the average value of 6.44 g. was still in the lower range of normal.

The average blood sodium levels were 349 mg. per 100 ml. in the prodromal phase, rising to 359 mg. during the early stages of an attack and returning to a normal figure of 333 mg. during recovery. In the interim phase it rose to 339 mg. In some cases we have observed a large diuresis accompanied by a large output of sodium at the termination of a sharp attack of migraine. In the recovery the blood sodium appeared to return to a normal level before the plasma proteins did so. The exact balance of these changes during an attack of migraine is the subject of further investigations.

It may be noted here that Duncan (1947) gives five conditions in which the blood sodium and chloride are raised. (a) Excessive administration of salt in cases of urinary suppression or oliguria; (b) the nephrotic stage of glomerulonephritis; (c) Cushing's syndrome; (d) congestive heart failure; and (e) excessive sweating with simultaneous restriction of fluid intake.

We have already mentioned that retention of sodium and chloride may also occur in women during the premenstrual phase.

The findings made in the first group of experiments might be explained theoretically in terms of the work demanded of the kidneys under conditions of water diuresis.

If the level of sodium in the blood is raised as a result of adrenocortical overactivity the cells of the renal tubules would have a greater amount of work to do in returning the sodium from the glomerular filtrate to the body fluids than if the blood sodium were at a lower level. This work would be further increased if the colloid osmotic pressure of the blood were low. Newburgh and Leaf (1950) have pointed out that renal work increases as the daily urinary volume rises (at levels above 2 litres a day), and that it is further increased if the urinary sodium chloride concentration is low.

If all these factors be borne in mind it is evident that during the pre-migraine state of a high blood sodium and a low colloid osmotic pressure the kidneys have an abnormally heavy task imposed upon them in conditions of water diuresis as compared with normal control subjects, and that as a result there is an escape of sodium. The greatest loss occurs early in the diuresis at the time of the maximum rate of urinary excretion.

In view of the great mental and physical activity of migrainous patients it is reasonable to suggest that they suffer from overactivity of the adrenal glands, including an oversecretion of adrenaline and adrenocortical hormones. The latter would account for the predisposition to maintain a high blood sodium before an attack of migraine, which is so often brought on by stress. Verney (1946) has shown in some experimental work on dogs that the production of the antidiuretic hormone is stimulated both by emotional stress and by a rise in the sodium chloride concentration in the arterial blood. In the migraine syndrome there is an analogous state, in which both factors, acting together, may give rise to water retention. The attack of migraine so caused is a physiological crisis which usually terminates by water diuresis and the excretion of the surplus sodium, at the moment when the secretion of the antidiuretic hormone fails. If this is the true course of events, the beneficial effect of urea may be understood to be due to the continuous mild diuresis which prevents the accumulation of sodium and of water in the blood.

In women, the particular influence of the sex hormones during the premenstrual phase may exacerbate the rise in blood sodium and chloride, so that an attack will almost inevitably occur at this time.

Conclusion

Migraine is a disease which is accompanied by a disturbance in salt and water metabolism. In the pre-migraine phase, and more particularly in the early stages of the attack itself, the blood sodium rises to a high level, while at the same time there is marked hydration of the blood. The excessive secretion of sodium and chloride which follows a water imbibition test suggests that an abnormal sodium metabolism is present almost continuously.

The suggestion is made that these disturbances are due to the disordered functions of the hormones governing salt and water metabolism of the adrenal cortex and posterior pituitary gland, and that in women they are exacerbated by the influence of certain sex hormones.

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occurred in the years 1898-1900. The occurrence of a modal age for the prevalence of deafness was most evident in New South Wales. The numbers of deaf-mutes enumerated at the three censuses in New South Wales are given by age and sex in Table I. The numbers

TABLE I.—*The Deaf-mutes in New South Wales as Enumerated at the Three Censuses*

Age Group	1911 Census		1921 Census		1933 Census	
	Males	Females	Males	Females	Males	Females
0-	8	8	7	10	7	4
5-	34	25	39	33	57	38
10-	(54)	(57)	43	43	56	33
15-	35	29	30	27	68	73
20-	36	29	(69)	(46)	49	49
25-	34	26	36	23	44	25
30-	24	30	35	32	(78)	(62)
35-	35	22	35	27	44	27
40-	14	22	22	25	31	28
45-	19	13	25	25	29	24
50-	13	15	9	17	28	18
55-	8	6	13	11	18	22
60-	11	7	12	14	15	14
65+	4	16	21	29	14	25
Unclassified ..	1	5	0	3	1	1
Total	330	310	396	365	539	443

in the modal age groups are given in parentheses. In 1911 the maximum prevalence of deaf-mutism occurred in the age group 10-14 years, and the statistician, G. H. Knibbs (1917), was inclined to ascribe the maximum to the more complete enumeration of the deaf at the school ages. In 1921, however, it was clear that this explanation was inadequate, for the maximum was to be found at the age group 20-24 years, and this maximum was due to the same "cohort" or group of persons, who had merely aged in between the censuses. The statistician, C. H. Wickens (1927), considered the possibilities of epidemic disease as a cause of the deafness in the following words: "The age incidence of the deaf-mutism varies in such a manner that it is apparent that the cause of the disability is of variable intensity, and there is some evidence to suggest that the increase in incidence of deaf-mutism at certain ages synchronizes with the occurrence of epidemic diseases, such as scarlet fever, diphtheria, measles, and whooping-cough." At the report on the census of 1933 the statistician, Roland Wilson (1942), again discussed infective disease as the cause of the deafness. We have followed the lead given by these census reports and have determined the date of birth of the deaf admitted to certain of the institutions throughout Australia. Here only the data from the New South Wales Institution for the Deaf and Dumb and the Blind will be discussed in detail, but similar considerations will apply to the data from the other states, which are also presented in the tables.

Births of the Deaf in New South Wales

Since the opening in 1861 of the New South Wales Institution for the Deaf and Dumb and the Blind there has been a well-kept register of the admissions. For the earlier admissions the month of birth was not always noted. We have been able to distinguish the sex, wherever it has not been explicitly stated, by means of the Christian name of the patient. In Table II the numbers of those born deaf admitted to institutions are given for New South Wales and other states. Since in the earlier years the admissions were few in number and did not seem to suggest epidemic occurrences, the results have been pooled up to the year 1880. From this year onwards the numbers of births of the deaf,

DEAFNESS AS AN EPIDEMIC DISEASE IN AUSTRALIA

A NOTE ON CENSUS AND INSTITUTIONAL DATA

BY

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It has generally been assumed that the epidemics of deafness and other congenital defects associated with births in the years 1938-41 and reported by Gregg (1941), Swan *et al.* (1943), Patrick (1948), and others from the various Australian states were a new and unusual manifestation of rubella. So far as we are aware there has been no report of an extensive epidemic, prior to these years, giving rise to cases of deafness. It is proposed to put on record here certain observations suggesting that similar epidemics have occurred in the past, notably a very extensive epidemic of births of the deaf in 1899 in the states of Australia. We have definite knowledge of a great epidemic of rubella throughout Australasia in the previous year. It is reasonable to presume provisionally that many of the cases of deafness were due to that disease, although it is not possible to relate individual cases of deafness to a history of maternal rubella.

Epidemic Nature of some Forms of Deafness

In the three successive Australian censuses of 1911, 1921, and 1933 the statistician's report on the census gave a chapter to the answers on the questions regarding blindness and deaf-mutism in the census. It was apparent that the prevalence of blindness rose with increasing age in the population. On the other hand, the prevalence of deaf-mutism showed a maximum in each census corresponding in each case to persons born in the years 1896-1900. The fact that 12 years elapsed between two of the censuses makes it possible to deduce that the excess of births of these deaf may have