The Exciting Causes of Epilepsy.

I have attempted to show that the predisposing cause of epilepsy is, in the majority of cases, an inherited neuropathic disposition, and that epilepsy may arise independently of any exciting or determining cause, being a spontaneous development attributable to the normal processes of brain growth and sexual maturation in predisposed persons.

In a number of cases, however, some exciting influence is at work in the production of the first epileptic seizure. It is a cardinal principle that the cause of epilepsy is that circumstance to which the first fit is apparently due. The instability of the brain, once induced, becomes a characteristic feature, so that further attacks recur quite independently of the original or any other obvious exciting cause.

The common period for the onset of epileptic fits is puberty, and in girls its onset is frequently accompanied by irregularity in the catamenia. There is little or no evidence to support a popular view that the regular establishment of the monthly period is attended by arrest of the seizures in those in whom the disease has arisen a few years earlier. The relation of epileptic fits to menstruation is well established. In most cases the seizures occur immediately before or shortly after the period, and in chronic cases the monthly period is characterized by an increase in their frequency.

The normal process of pregnancy has also some influence in the causation of epilepsy in women. We find that pregnancy may be the exciting cause of the disease in some cases, that it may induce a relapse of the seizures in cases in which a cure has apparently been established or in which a long remission has occurred. On the other hand, pregnancy has been known to produce a temporary arrest or amelioration of fits, and there are well-authenticated cases in which a permanent arrest has been brought about in this way. Accouchement and the puerperium have also an important bearing upon epileptic fits. I have seen a number of cases in which the disease clearly originated at this time in the form of a puerperal eclampsia and persisted for many years as confirmed epilepsy. These cases are interesting, as they argue in favour of the view originally propounded by Féré—that some types of puerperal eclampsia are merely the onset of epilepsy in an acute form in predisposed persons.

Sleep is another physiological condition eminently favourable to the onset of epileptic fits. In many cases fits only occur during sleep, so that a clinical type has been recognized as nocturnal epilepsy. There can be no doubt that cases of nocturnal epilepsy may exist unrecognized for many years, until a convulsion in the daytime establishes the existence of the disease. Voisin mentions the following phenomena as suggestive of the nocturnal form: Intermittent wetting of the bed, occasional periodic and temporary mental dullness and irritability, well-marked lapses of memory, somnambulism, and unexplainable outbursts of temper. Pick has called attention to the frequency of fits during the hours of deepest sleep—namely, during the first hour or two after falling off to sleep, and again before waking. My own observations show that the greatest number of fits occur during the hour or two after falling off to sleep—a period which probably coincides with the greatest degree of cerebral anaemia. Whether these facts are to be explained on the theories of brain anaemia (Pick), of lessened inhibitory control (Clark), of the alkaline tide (Haig), or of increase in the rate of blood coagulation (Turner),¹⁰ are points on which evidence is insufficient.

A second and smaller group of epilepsies is found in which a definite extraneous cause acts as the excitant of the first fit, and, as such, of the disease. These causes may be:

1. *Psychical influences*, such as emotional excitement, fear, shock, anxiety, grief, or overwork. Gowers, in particular, regards this as the most potent of all the immediate causes of epilepsy. It was the ascribed cause in only 4 per cent. of my cases, being twice as common in females as in males. In most of the cases a strong neuropathic predisposition was found.

2. *Infective diseases*. The most frequent is scarlet fever, but measles, pertussis, influenza, and diphtheria, have also been observed. An interesting feature in this connexion is the occasional temporary arrest of the fits in consequence

of an attack of pyrexia, probably due to the arrest in the chloride excretion with corresponding retention of bromide salts (Hoppe).

3. *Reflex causes* play an important part as excitants of epilepsy, more especially in children and young adults predisposed by heredity to nervous instability. I would especially call attention to the necessity of a careful examination of the nose (for adenoids, polypi, or foreign bodies), of the eyes (for errors of refraction), of the ears (for chronic otorrhoea), of the teeth, the genital organs, the stomach, and of the intestine (for the presence of worms).

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OPTIC NEURITIS AND SUPPURATIVE OTITIS.

A FURTHER INVESTIGATION INTO THE FREQUENCY AND SIGNIFICANCE OF OPTIC NEURITIS AND OTHER VASCULAR CHANGES IN THE RETINAE OF PATIENTS SUFFERING FROM PURULENT DISEASE OF THE MIDDLE EAR.

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A PAPER by us on this subject, giving the results of the examination of the eyes and ears in 100 consecutive cases of purulent middle-ear disease, appeared in the *BRITISH MEDICAL JOURNAL* of November 23rd, 1907. Since then we have continued this investigation, and examined a second series consisting of 60 cases, and the results of these, along with those of our first 100 cases, are embodied in the present article; the conclusions arrived at, therefore, are founded upon the examination of the eyes and ears of 160 cases. Where possible, each individual case has been kept under observation until the termination of the ear affection in recovery, either as the result of local treatment or after operation. Of the 160 cases, 100 presented themselves after an interval of several months for a second ophthalmoscopic examination, and quite a number were seen three or four times.

We desire to emphasize the special importance, when examining the eyes in this class of case, of duly considering the effects of age, occupation, eye strain, and errors of refraction. These considerations constituted at first the greatest difficulties we had to contend with, and explain the large residue of 46 doubtful cases. A wider experience of the types met with has to a large extent obviated these difficulties.

The plan of this paper is, first, to give the results of the examination of the second series of 60 cases, comparison being made, in tabulated form, with those of the first series of 100 cases; secondly, to present the results obtained from the total number (160 cases); thirdly, to discuss one or two of the more interesting problems in connexion with optic neuritis of otitic origin; and finally, to state briefly the conclusions arrived at as the result of our investigations.

I.—THE EXAMINATION OF THE SECOND SERIES (60 CASES).

As in our first series of cases, the aural examination included: (1) A history of the ear affection; (2) the testing of the hearing distance by a watch normally heard at 36 inches; (3) the application of Rinne's and Weber's tests when the age of the patient permitted, a tuning-fork

Sex.	Age.	Cause.	Symptoms.	Ear Affected.
Males ... 36	Under 15 years ... 18	Scarlet fever 10	Otorrhoea 60 cases	Both ears ... 5 cases.
Females ... 24	Between 15 and 30 ... 29	Measles 11	Impaired hearing ... 60 "	Right ear ... 29 "
	Over 30... .. 13	Whooping-cough ... 1	Earache 21 "	Left ear ... 26 "
		Pneumonia 3	Tinnitus 23 "	
60	60	Cold 3	Giddiness {slight ... 9 "	65 affected ears.
		Sore throat 2	" {marked ... 9 "	
		Burn 1	Headache {slight ... 17 "	Evidences of past suppuration
			" {marked ... 21 "	in 31 ears.
		29		
<hr/>				
Hearing.		Granulations or polypi	Present in 33 ears	Tuning-fork Tests.
2 inches and under ...	40 ears	Cholesteatoma	10 "	In 56 discharging ears, Rinne
Above 2 in. and under 12 in.	19 "	Perforation in membrana flaccida	6 "	was negative in 54. In 46 cases of
12 in. and over	5 "		49	unilateral suppuration, Weber was
Too young	1 "			referred to the affected side in 34,
	65	Chronic otitis media purulenta in	63 ears	was indefinite in 6, and referred to
		Acute " " "	2 "	the unaffected side in 6.

TABLE II.—REFRACTION OF THE 100 CASES (FIRST SERIES).

Examination of 200 Eyes: 108 Emmetropic (Both Eyes), 3 (One Eye).

The Refraction of the Ametropic Eyes was as follows:

Hypermetr.	Hypermetr. Astig.	Myop.	Myop. Astig.	Mixed Astig.	Refraction not Stated.	Emmetropic ... 117 Ametropic ... 87 Not given ... 2
26	27	10	5	19	2, i.e., one case	Total ... 200

REFRACTION OF THE 60 CASES (SECOND SERIES).

Examination of 120 Eyes: 68 Emmetropic (Both Eyes), 7 (One Eye).

Hypermetr.	Hypermetr. Astig.	Myop.	Myop. Astig.	Mixed Astig.	Refraction not Stated.	Emmetropic ... 55 Ametropic ... 45
12	10	8	8	7	0	Total ... 120

TABLE III.

Class I.—A. 100 Cases. Results of First Eye Examination (First Series).

Division I.—Emmetropics ... 54	Number showing vascular changes:— 39, or 72.2 per cent.	Normal. ... 15
Division II.—Ametropics ... 46	33, or 71.7 per cent.	13
100	72, or 72 per cent.	28

B. 60 Cases. Results of First Eye Examination (Second Series).

Division I.—Emmetropics ... 34	16, or 47.0 per cent.	18
Division II.—Ametropics ... 26	8, or 30.7 per cent.	18
60	24, or 38.8 per cent.	36

Class II.—A. 60 Cases. Examined Twice—Ears and Eyes (First Series).

	No.	Normal.	First Examination of Eyes (vascular changes).	Second Examination of Eyes.	Second Examination of Ears.
Division I.—Emmetropics	34	7	27	{ 14 unchanged { 2 improved { 11 worse	{ 10 unchanged or worse. { 4 improved. { 1 healed for six months. { 1 no change. { 9 unchanged or worse. { 2 improved.
Division II.—Ametropics	26	6	20	{ 13 unchanged { 4 improved { 3 worse	{ 9 unchanged or worse. { 4 improved. { 1 improved. { 1 unchanged. { 3 unchanged or worse.
	60	13	47		

B.—40 Cases. Examined Twice—Ears and Eyes (Second Series).

Division I.—Emmetropics	20	8	12	{ 6 unchanged { 3 improved { 2 worse	{ 4 unchanged or worse. { 1 improved. { 1 radical. { 1 improved. { 1 unchanged. { 1 radical. { 2 unchanged or worse.
Division II.—Ametropics	20	12	8	{ 3 unchanged { 3 improved { 2 worse	{ 1 radical. { 2 unchanged or worse. { 1 radical. { 2 unchanged. { 1 unchanged. { 1 worse.
	40	20	20		

TABLE IV.
First Series.

A.—Cases showing distinct vascular engorgement at the First Examination: 20	Progress of the Ear condition subsequent to the First Examination: { 15 per cent. healed or improved. { 85 per cent. unchanged or required radical.
B.—Cases showing normal fundi at First Examination: 13	{ 61.5 per cent. healed or much improved. { 38.4 per cent. unchanged or required radical. NOTE.—In 40 per cent. of the 38.4 per cent. the treatment was completely neglected.
Second Series.	
A.—Cases showing distinct vascular engorgement at the First Examination: 12	Progress of the Ear condition subsequent to the First Examination: { 16.6 per cent. healed or improved. { 83.4 per cent. unchanged or required radical.
B.—Cases showing normal fundi at First Examination: 20	{ 60 per cent. healed or improved. { 40 per cent. unchanged.

TABLE V.
First Series.

EYE.	EAR.
Condition of the fundi at the Second Examination compared with the condition of the First Examination:	Progress of the Ear condition as shown at the Second Examination:
27 unchanged	{ 19 unchanged or worse. 8 improved or healed.
6 improved	{ 3 improved or healed. 3 unchanged.
14 worse	{ 12 unchanged or worse. 2 improved.
Second Series.	
9 unchanged	{ 6 unchanged or worse. 2 improved (1 after radical).
6 improved	{ 3 unchanged. 3 improved (2 after radical).
4 worse	4 unchanged or worse.

TABLE VI.
160 Cases. Results of First Eye Examination (Combined Series).

	No.	Normal.	Results of First Eye Examination (vascular changes).
Division I.—Emmetropics ...	88	33	55=62.5 per cent.
Division II. Ametropics ...	72	31	41=56.9 per cent.
	160	64	96

TABLE VII.

	No.	Normal.	First Examination of Eyes (vascular changes).	Second Examination of Eyes.	Second Examination of Ears.
Division I.—Emmetropics	54	15	39*	{ 20 unchanged 5 improved 13 worse	{ 14 unchanged or worse. 6 improved (1 after radical) 3 healed (1 after radical). 2 unchanged. 11 unchanged or worse. 2 improved.
Division II.—Ametropics	46	18	28	{ 16 unchanged 7 improved 5 worse	{ 11 unchanged or worse. 5 improved (1 after radical) 3 improved (1 after radical). 4 unchanged. 5 unchanged or worse.
	100	33	66		

* One case has been withdrawn from this list, as owing to defective intelligence no dependence could be placed upon the results of the examination. Vascular engorgement was present in the left eye.

TABLE VIII.

A.—Cases showing distinct vascular engorgement at First Examination:	Progress of the ear condition subsequent to the First Examination:
32	15.8 per cent. healed or improved.
B.—Cases showing normal fundi at First Examination:	
33	60.75 per cent. healed or improved.

TABLE IX.

SECOND EXAMINATION OF EYES.	SECOND EXAMINATION OF EARS.
36 unchanged ...	{ 25 unchanged or worse. 11 improved (2 after radical).
12 improved ...	{ 6 healed or improved (2 after radical). 6 unchanged.
18 worse ...	{ 16 unchanged or worse. 2 improved.
66	

In addition to these 11 cases of definite optic neuritis, there were at the first examination 39 showing vascular changes in the fundus, not actually amounting to well-marked optic neuritis, but of such a pronounced character that they could not be regarded as normal, even when due consideration was given to possible extraneous causes, such as age, occupation, error of refraction, and straining of the eyes. There remained 64 cases in which both fundi were unquestionably normal, and a number of doubtful

cases, 46 in all, in which the changes were slight or could probably be accounted for by other conditions.

In Table VIII a comparison is made between the progress of the ear condition in 32 cases which showed the most marked vascular changes in the retinae, and 33 in which the fundus was normal. The striking fact is brought out that in cases of purulent middle-ear disease, in which the fundus is normal, the prospects of recovery without operation are four times greater than when

distinct evidences of vascular engorgement of the fundus are present.

The relationship between the progress of the eye and of the ear conditions is brought out in Table IX, which contains 66 cases, examined more than once, showing evidences of vascular engorgement at the first examination. It will be seen that there has been proportionately a much greater improvement in the aural condition (50 per cent.) in those cases in which the eyes have shown a corresponding change for the better than in those in which the vascular engorgement has become still more marked; in these last only 11.2 per cent. showed improvement. This fact is of distinct prognostic value, showing that with improvement in the condition of the fundus a favourable termination to the aural disease may be more hopefully looked for than when the eye condition remains *in statu quo* or becomes worse.

In 125 of the 160 cases one ear only was affected. In 21 of these the evidences of vascular engorgement were confined to or more marked in one eye; in 14 to the eye of the affected side, and in 7 to the eye of the unaffected side. In all the other cases—104 in number—the ocular condition was equally marked in both eyes, or the fundi were normal. It may thus be said, without, however, attaching too much importance to the point, that there is a greater tendency for the eye on the same side as the ear affection to show vascular changes. The following illustrative case may be cited:

A girl aged 13 had discharge from the left ear for eight months following pneumonia and from the right ear for three weeks; there were also frequent headaches. In the left ear was a large perforation, involving the lower half of the membrane, and in the right a small central perforation with scanty discharge. Both eyes were emmetropic, and a slight degree of optic neuritis was found in both, but more marked in the left. Four months later the right ear (the side on which the retina was least affected) was found dry, while the left ear (the side on which the optic neuritis was most marked) showed no improvement. The optic neuritis in the left eye had become distinctly more marked, while in the right the change, though slight, was in the direction of improvement.

Friedenberg,² in an interesting and instructive article, speaks of the value of ocular changes confined to or more marked in one eye, in the following sentence: "The more pronounced development on one side of the optic nerve involvement generally has no special meaning; in sinus thrombosis, however, value may be attached to it." In the course of his article Friedenberg quotes a case of sigmoid sinus thrombosis reported by Grüning in which, after the mastoid cells had been opened and the sinus exposed on both sides, there were no external indications to show on which side the sinus was affected. In both eyes there was vascular engorgement, which in the left, however, rapidly progressed to definite optic neuritis. For this reason alone the left sinus was opened and the left internal jugular vein resected, when a septic thrombus was discovered and removed; complete recovery followed.

The type of optic neuritis met with in this investigation has been usually mild; in 4 cases it was pronounced, and to them the term "choked disc" could be applied. The vision, however, in so far as could be ascertained, has remained unaffected and there has been no true secondary atrophy. In regard to this last point we wish to emphasize the fact that the optic neuritis associated with pyogenic intracranial lesions of otitic origin need not be followed by atrophy and blindness, differing in this very important respect from the optic neuritis found in connexion with tumours of the brain. Sir Victor Horsley³ says in regard to optic neuritis resulting from intracranial disease:

Therefore it is now possible . . . to say that in no case of optic neuritis (not of course of toxic or anaemic origin) should the process be allowed to continue after it has once been diagnosed, and that if blindness results therefrom the responsibility is very heavy on any one who fails to advise such a simple proceeding as opening the dura mater.

Two cases of otitic perisinus abscess with paralysis of the sixth cranial nerve and double optic neuritis (choked disc) reported by one of us⁴ (J. S. B.) demonstrate that optic atrophy does not necessarily follow optic neuritis of otitic origin, even when the dura mater is not opened. Dr. Rowan, who repeatedly examined these two cases when in the acute stage, again examined them on March 25th, 1909, three years and one and a half years respectively after the disappearance of the neuritis. In each case the vision was normal (§ J. 1), as were also the visual and colour fields, as tested by perimeter. In neither

case was the dura mater incised. In the light of these and other cases it seems to us that Sir Victor Horsley's dictum might require some modification, or, at any rate, further investigation.

THE CAUSATION OF OPTIC NEURITIS IN CONNEXION WITH PURULENT EAR DISEASE.

In discussing the question of how these changes in the fundus of the eye are brought about, the great difficulty is the absence of pathological evidence based upon *post-mortem* examinations. In gross inflammatory intracranial lesions, such as lepto-meningitis, brain abscesses, etc., where information from *post-mortem* examinations is obtainable, the most generally accepted view is that increased intracranial pressure plays an important part in the causation of the accompanying optic neuritis. May not intracranial pressure be responsible also for those vascular changes in the eyes in certain cases of apparently simple temporal bone suppuration?

We know that serous meningitis is a not uncommon complication of purulent middle-ear disease. This form of meningitis, when present in a marked degree, may simulate, as regards symptoms, either purulent lepto-meningitis or brain abscess, but it is well known to present more frequently than either of these definite vascular changes in the fundus amounting in most instances to optic neuritis. In fact, judging from published reports of undoubted cases of this affection, optic neuritis is present in a very large majority. This is recognized by such an eminent authority as Jansen.⁵ We think we have good grounds for believing that milder forms of serous meningitis occur much more frequently during the course of a chronic middle-ear suppuration than has hitherto been supposed.

We have been impressed, on questioning patients closely, with the frequency of symptoms pointing to the probability of meningeal attacks, such as frequent bouts of headache accompanied in some with nausea, vomiting, or giddiness, and in others with elevated temperature. In over 50 per cent. of our cases headache was complained of. Several of our patients were compelled to lie in bed for a week or even longer during these attacks, and we have also been informed by several parents that their children have been repeatedly sent home from school on account of severe headache and sickness. It may be postulated that in a patient suffering from purulent ear disease these symptoms coming on periodically are highly suggestive of meningeal irritation, probably of the nature of mild serous meningitis.

It is difficult to explain how comparatively mild attacks of serous meningitis may bring about vascular changes in the fundus of the eye. It is probable, however, that localization has, as in other intracranial lesions, an important bearing on the causation of these changes. In marked serous meningitis with very pronounced symptoms the fluid, large in amount and under considerable pressure, is widely distributed over the base of the brain. On the other hand, in the mild type of which we are speaking, it is reasonable to suppose that the serous effusion is more localized; constituting, we may say, abortive attacks cut short for some reason before becoming widely spread. For example, the effusion may take place in those well-known spaces, the subarachnoid cisterns at the base of the brain, and one or other of them may become a reservoir in which the effused fluid collects.

Supposing the anterior cisterna, or even its anterior half where lie the optic chiasma and the commencements of the optic nerves, carrying with them their vaginal sheaths, be the region in which the excess of fluid collects, vascular changes in the fundus amounting even to well-marked optic neuritis may reasonably be supposed to result. No doubt every one is familiar with cases of fatal lepto-meningitis in which the condition has been mainly limited to the anterior cisterna. While believing that pressure exercised in this way has probably a great deal to do with the causation of the type of optic neuritis under consideration, there are at least two modifying influences at work: (1) there may in some individuals be conditions, anatomical or constitutional, specially favourable or otherwise to the onset of optic neuritis—in a word, idiosyncrasy; and (2) the chemical character of the fluid exercising pressure upon such delicate structures as the optic nerves is of importance. In serous meningitis the fluid, while no doubt sterile in so far as pus-forming organisms are concerned, is presumably not free from their toxic products. A very

limited amount of pressure, therefore, may be quite sufficient in some individuals to excite changes in the optic nerves, when the evil effect of the pressure is enhanced by the chemical irritation of the toxins.

We venture to suggest that in many cases of purulent thrombosis of the large sinuses, and in at least a certain proportion of the cases of brain abscess, optic neuritis, when present, is caused by serous meningitis, either accompanying or immediately preceding these grave complications of purulent ear disease.

CONCLUSIONS.

Briefly stated, the following are the conclusions arrived at as the result of this investigation:

1. Optic neuritis may occur in cases of purulent middle-ear disease without obvious signs of an intracranial complication (11 times in 160 cases, or 6.8 per cent.).
2. Apart from optic neuritis, vascular changes of a lesser degree are frequent (39 times in 160 cases, or about 25 per cent.).
3. Cases of purulent middle-ear disease, in which optic neuritis or vascular engorgement of the fundus is present, are much less amenable to local treatment than those in which the fundus is normal.
4. As a general rule, an improvement in the eye condition is accompanied by improvement in the aural condition, while an increase in the intensity of the changes in the fundus or their persistence is associated with less amenability to treatment and greater gravity of the ear condition.
5. The most probable cause of vascular engorgement of the fundus or optic neuritis is serous meningitis (diffuse or localized).
6. Optic neuritis caused in this way is not usually followed by atrophy, and unless there are other symptoms demanding it, opening of the dura mater is unnecessary.

The practical lessons deducible from these are (1) that a case showing these changes in the fundus should be closely watched, and their existence regarded as an additional reason for the early performance of the radical mastoid operation; (2) if, on the other hand, they show a tendency to clear off, especially with improvement in the ear condition, or if the fundus is normal to begin with and remains so, we may with more confidence look for a favourable response to conservative treatment.

Finally, we again plead for a more systematic general use of the ophthalmoscope in cases of purulent disease of the middle ear, as we are convinced that the trouble involved will be more than repaid by the results, both as regards the progress and prognosis of this disease.

NOTE.—Since writing this we have had the opportunity of reading a most interesting paper⁶ on experimentally-induced choked disc, by Drs. Harvey Cushing and James Bordley. From an experimental standpoint these two observers come to conclusions very similar to our own, and we quote them here in full:

1. That the occurrence of neuro-retinal oedema is primarily dependent on the passage of cerebro-spinal fluid under tension from the subarachnoid space of the interpeduncular region (anterior cisterna) into the vaginal sheath of the optic nerve, and that cerebral decompression often allows the process to subside, owing to a resultant diminution of tension from release of the confined fluid.

2. That the experimental work corroborates many of the more recent clinical observations in showing that a choked disc, even of considerable height, may be rapid in its formation and, provided it has not gone on to the stage of new tissue formation, may rapidly subside, and thus speaks strongly in favour of a mechanical as opposed to a chemical or inflammatory origin for the lesion.

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THE Association of German Scientists and Medical Practitioners will hold its eighty-second meeting at Königsberg this year from September 18th to 24th. Among the addresses of medical interest to be delivered in full session are the following: Dr. Cramer, of Göttingen, "Puberty and the School"; and Professor von Monakoff, of Zurich, "Localization of the Functions of the Brain." The combined medical sections will hold eighteen sittings. Communications should be addressed to the Managing Committee, Drummstrasse 25-29, Königsberg, Prussia.

A SHORT NOTE ON THE SCOPOLAMINE-MORPHINE NARCOSIS.

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IN the course of some pharmacological experiments with scopolamine and morphine I found in animals certain effects produced on the respiration similar to those which sometimes attend the clinical use of scopolamine and morphine as an anaesthetic.

The experiments were made on cats, the blood pressure and respiratory movements being recorded in the usual way.

The preparation used was the tablet form of hyoscine hydrobromide (gr. $\frac{1}{100}$) and morphine sulphate (gr. $\frac{1}{2}$), prepared by Duncan, Flockhart and Co., Edinburgh. This dose was calculated as being that for an adult of 70 kg., giving a dose per kilogram of body weight of hyoscine gr. $\frac{1}{7000}$, morphine gr. $\frac{1}{1400}$. Accurate dosage was attained by dissolving one tablet in a definite quantity of Ringer's solution, and using intravenously such proportion of the solution as was required.

Fig. 1 (cat 2,850 gram) shows the normal respiratory

Fig. 1.

and circulatory movements, and the effects thereon of a normal therapeutic dose of hyoscine and morphine intravenously. There is no immediate effect on the respiration rate, but ten minutes later, as shown in Fig. 2,

Fig. 2.

the respiratory rate has been reduced from 34 to 20 a minute.

Fig. 3 shows the effect of a second intravenous injection,

Fig. 3.

given thirty minutes after the first, the dose of hyoscine and morphine being this time equal to only half the previous dose. Yet one notices the immediate reduction in the respiratory-rate of from 26 to 14 a minute, and later to 10 a minute, as shown in Fig. 4, taken three minutes later. Thirty minutes later, when the animal was killed, the respiration-rate was 16 a minute.

In the next experiment a cat of 3,900 grams was taken, and to it was given thrice the normal therapeutic dose