The doctor's pocket remembrancer / J.C. Eno Ltd.

Contributors

J. C. Eno Ltd

Publication/Creation

London : J.C. Eno, 1931.

Persistent URL

https://wellcomecollection.org/works/cetsk9us

License and attribution

Wellcome Collection
183 Euston Road
London NW1 2BE UK
T +44 (0)20 7611 8722
E library@wellcomecollection.org
https://wellcomecollection.org

The
DOCTOR'S POCKET
REMEMBRANCER

The

DOCTOR'S POCKET REMEMBRANCER

LONDON:
J. C. ENO LTD.
1931

THE ENO MEDICAL REMINDERS

No. 1. The Doctor's Pocket Remembrancer.

No. 2. The Practitioner's Pocket Book.

No. 3. The Panel Doctor's Pocket Book.

No. 4. The Doctor's Emergency Reminder.

No. 5. Urgent Abdominal Diagnostics.

TABLE *of* CONTENTS

	PAGE
REFLEXES	14
Babinski's Sign	14
Knee-Jerk	15
Ankle-Clonus	16
MUSCULAR ATROPHY ..	18
Primary muscular dystrophies	18
Acute anterior polio-myelitis	18
Tooth's peroneal type of progressive muscular atrophy	19
Peripheral neuritis	19
Progressive muscular atrophy	19
Amyotrophic lateral sclerosis	20
Syringo-myelia	20
Transverse myelitis	20
Injury to peripheral nerves	21
SPASTIC PARAPLEGIAS ..	22
Disseminated sclerosis ..	23
Ataxic paraplegia	24
Cerebellar abscess and tumour	24
Transverse myelitis	24
Primary lateral sclerosis ..	25
Amyotrophic lateral sclerosis	25
Syringo-myelia	26
Hæmorrhage into the cord ..	26
Meningitis	26
Bilateral cerebral hæmorrhage or softening	26
FLACCID PARAPLEGIAS ..	27
Acute anterior polio-myelitis	27

PAGE

Tooth's peroneal type of progressive muscular atrophy 27
Peripheral neuritis in children 28
Peripheral neuritis in adults 28
Transverse myelitis 29

PARAPLEGIAS NOT STRICTLY OF SPASTIC OR FLACCID TYPE .. 30
Pseudo-hypertrophic muscular paralysis 30
Primary muscular dystrophy 31
Landry's paralysis 31
Tabes dorsalis 31
G.P.I. 32
Functional paraplegia .. 32

ATAXY 33

TREMORS 35
Paralysis agitans 35
General paralysis of the insane 36
Graves' disease 36
Lead poisoning 36
Alcoholism 36
Chronic hemiplegia .. 37
Disseminated sclerosis .. 37
Various ataxias 37

COMA 38
Cerebral hæmorrhage .. 38
Pontine hæmorrhage .. 38
Cerebral embolism 39
Opium poisoning 39

PAGE

Carbon monoxide 39
Alcohol, cold and heat stroke 39
Epileptic coma 40

A FEW EYE MEMORANDA 41

Acute Inflammation .. 41
Failing Vision 42

DEAFNESS 44
Rinné's Test 44
Weber's Test.. 44

GENERAL ABDOMINAL
DISTENSION 46
Ascites 46
Phantom Tumour 47
Distended Bladder 48
Ovarian Cyst 48
Tympanites 48

JAUNDICE 49
Catarrhal jaundice 49
Malignant disease 49
Gall stones 49
Chronic pancreatitis .. 49
Cirrhosis 50
Syphilis of the liver .. 51

A FEW URINE POINTS .. 52
Sugar in Urine 52
Legal's Test for Acetone .. 52
Hæmaturia 53
Acetic Acid Test for Albumen 54

IN this little book for the waistcoat pocket it would, of course, be impossible, even ever so briefly, to epitomise the essentials of diagnosis. Every practitioner would be wise to keep on his handiest shelf a copy of French's *Index of Differential Diagnosis* and Leftwich's *Index of Symptoms*, two constantly useful books, to which few need be too proud to refer.

In this booklet are summarised a few simple reminders of points that sometimes slip the memory, or fail to present themselves to consciousness at the right moment. This is especially likely with the symptoms of diseases of the nervous system, to which this book is chiefly devoted. The art of diagnosis, like those of treatment and prognosis, is by no means an exact one. Elements of judgment and even of guessing enter into it constantly. Probability is often as near to truth as we can reach;

and even the wisest, most judicial, and most experienced err daily. Complacency and cocksureness are out of place in the physician, and the shrewdest and best-informed is usually the humblest. Nearly every diagnosis should be treated as provisional. Hence the folly of too much dogmatism either of diagnosis or of prognosis in communications to patients. These cannot possibly realise the difficulties of the problem, and, being unable to distinguish honesty from indecision, quickly lose confidence, to their own undoing. That which is certain may well be communicated; but, in conveying that which is not certain, the Delphic precedent may wisely be followed. Students and younger practitioners will perhaps forgive a few warnings :—

Beware of assuming neuroses because symptoms are indefinite

or not according to text-book classifications.

Be wary in diagnosing a disease which is in fashion, or one which you have recently been reading about. Symptoms can often be squeezed into a preconceived picture. They should make their own picture.

In diagnosing the cause of pain, recall your anatomy.

Cultivate the habit of saying to your patient nothing that need be revoked, even though your private diagnosis or prognosis may need altering. Keep a backdoor open, or you will often be unjustly cornered.

Remember that social position, unattractive looks, middle age and spinsterhood do not negative pregnancy. But this diagnosis should never be uttered until it is beyond all doubt.

Remember that nearly all serious, even fatal, disease has

very small beginnings. Never pooh-pooh apparently trifling symptoms.

Do not be too proud to use the stethoscope and the clinical thermometer; nor be in so much of a hurry as to neglect to examine the throat in children; or to examine the urine, test the reflexes and examine the optic disc, whenever these observations might conceivably help an accurate diagnosis.

Whenever malignant disease or tubercle is recognised or suspected, examine every other likely position for signs of the disease. At the same time, remember that abnormal physical signs in different parts of the body may be due to different and unrelated causes.

Let every question put to a patient be related to coherent thought. Random questions, yielding a succession of negative

answers, are not calculated to inspire confidence. Remember Voltaire: "A man should be judged by his questions rather than by his answers."

Listen to your patient's tale, but keep your mental salt-cellar handy. His misstatements may be informing and significant.

REFLEXES.

It should be remembered that a destructive lesion of the upper neuron tends to remove the normal inhibitions, and consequently to allow exaggerated reflex responses; and that irritative (but not destructive) lesions of the lower neuron tend to bring about the same result. Destructive lesions of the lower neuron, of course, tend to prevent reflex responses.

Babinski's Sign.—To test the plantar extensor reflex, ask the patient to lie on his side, with legs a little flexed at hip and knee, and with both feet turned out and resting comfortably on the bed. The ankle having been grasped by one hand, so that the whole foot may not be markedly flexed towards the shin, a penholder or pencil is firmly to be passed along the outer side of the sole from heel to toe. Babinski's sign consists in dorsal flexion reflex of the big toe instead

of its normal reflex of plantar flexion. This sign, or extensor plantar reflex, if present after the first few years of life, invariably indicates organic nervous disease. Its absence, however, does not negative this, for it is absent in anterior polio-myelitis, locomotor ataxy, Tooth's peroneal type of progressive muscular atrophy, and peripheral neuritis. It is practically always present in hemiplegia due to cerebral hæmorrhage, thrombosis, embolism or tumour, as well as in cerebellar tumour, transverse myelitis, disseminated sclerosis, lateral sclerosis, and Friedreich's Ataxy.

When Babinski's sign is present, the abdominal reflex (contraction of abdominal muscles when the abdomen is stroked lengthways) is absent, and it is present when Babinski's sign is absent.

Knee-Jerk.—Exaggerated knee-jerk, equal in both legs,

plantar reflex being flexor, generally indicates merely functional trouble associated with any form of ill-health. If, however, the plantar reflex is extensor, organic trouble is indicated, except in infancy. Loss of knee-jerk indicates organic disease, of which it may be the only sign. The presence of the knee-jerk does not, of course, negative organic nervous disease; since the reflexes concerned may not yet be affected.

Ankle-Clonus. — Lay the patient on his back with knees partially flexed. Then quickly, but without undue force, hold the foot by its outer part, keeping it well rotated outward, and flex it towards the shin. If ankle-clonus is present, a series of very rapid rhythmical contractions or jerks occurs at the ankle-joint, and these continue as long as the pressure on the sole is maintained. If jerks either do not occur or soon cease,

although the pressure is maintained, ankle-clonus is not present. Ankle-clonus is certain evidence of organic disease of the central nervous system. Exaggerated knee-jerk alone may be functional or organic.

MUSCULAR ATROPHY.

If the atrophy is merely part of a general wasting of the body, or is owing to disuse or to primary muscular dystrophies, there is no reaction of degeneration (R.D.) when the Faradic current is applied to the muscle, or the Faradic or galvanic current is applied to its nerve. This is to say, muscular contraction occurs as it does in health.

The *primary muscular dystrophies* may be recognised by their slow progressive course, by their insidious onset in children, and by their frequent occurrences in other members of the family. In all the following diseases, the reaction of degeneration occurs, partially or completely.

Acute anterior polio-myelitis (infantile paralysis) has an acute onset, and, wasting having reached a certain point, tends to improve

slightly and then to maintain a "status quo."

Tooth's peroneal type of progressive muscular atrophy frequently follows a febrile illness in childhood. Generally the first sign is inability to elevate the big toe, which hangs down. Very slowly the other muscles of the legs become affected, and ultimately the hands. There are no sensory disturbances.

In *peripheral neuritis*, the affected muscles are symmetrical. Superficial and deep reflexes are at first exaggerated, later disappearing. Slow recovery is usual. See p. 28.

Progressive muscular atrophy occurs in adult life. Its onset is insidious, the small muscles of the hands being first affected, resulting in "claw hands." Very slowly, the other muscles of the arm become affected, the affected muscles being supplied by different nerves—all those muscles in a given region

being affected before others also supplied by the same nerve. The symptoms are bilateral and symmetrical. Sensory disturbances are absent.

Amyotrophic lateral sclerosis presents the early signs of progressive muscular atrophy, but these are accompanied by spastic paralysis of the legs (without wasting), and exaggerated knee, ankle and plantar reflexes. Here also sensory disturbances are absent, and the onset is insidious.

In *syringo-myelia*, skin sensibility is normal, except that in certain variable areas pain cannot be distinguished from touch, or heat from cold.

A high *transverse myelitis* may cause symptoms resembling progressive muscular atrophy and amyotrophic lateral sclerosis; but sensation is generally affected, the onset is more or less sudden, and

the progress is not continuous, often ceasing if the patient lives.

Injury to peripheral nerves, *new growths* pressing on nerve-trunks, *gummata* and *sciatica* should be remembered as possible causes of wasting.

SPASTIC PARAPLEGIAS.

These result from lesions of the pyramidal tract between the higher part of the lumbar enlargement and the Rolandic cortex. They are generally marked by exaggerated tendon reflexes, by the presence of ankle-clonus and the extensor plantar reflex (Babinski's reflex), by the absence of muscle wasting apart from that due to disuse, by spasticity, and by the absence of R.D. (Reaction of degeneration). In young children, such causes as meningocele, myelocele, hydrocephalus, spina-bifida, congenital cortex defect, encephalitis and meningitis must be borne in mind. Physical signs and history will generally serve for their diagnosis. These infantile diplegias and congenital malformations having been excluded, *spinal caries* accounts for most of the spastic paraplegias of childhood.

Paraplegia from this cause is often accompanied with anæsthesia of the legs. *Friedreich's Ataxy* is a familial disease which generally shows itself between the ages of 6 and 15. Ataxia begins in the legs, and slowly and insidiously progresses to the arms and body. Ultimately paraplegia results. There are no sensory disturbances. Knee-jerks are absent. Generally some deformity results. The big toe is often fixed in an upwardly flexed position (Hallux erectus).

In adult life, the chief causes of spastic paralysis are the following :—

Disseminated sclerosis generally first appears between the ages of 20 and 40. It is marked by intention tremor (generally most marked in the arms, the head and neck being unaffected), the common signs of spastic paraplegias, nystagmus, a spastic or ataxic gait, exaggerated knee-jerks, ankle-

clonus, and slow, staccato, scanning speech. Generally a central scotoma exists. The subjective sensory signs are much more marked than the objective ones. Romberg's sign (see p. 33) is absent. The disease is not easy to recognise in its early stages, as few signs may show themselves, and these may constantly vary.

Ataxic paraplegia generally attacks men in middle age. Its symptoms much resemble those of disseminated sclerosis, but ataxy, nystagmus, and speech-changes are absent. There is no anæsthesia.

Cerebellar abscess and tumour even more closely simulate disseminated sclerosis, causing intention tremors in the hands, hesitant speech, etc.; but the addition of double optic neuritis, headache and vomiting indicates cerebellar trouble.

Transverse myelitis may be primary, or due to pressure by

aneurism, new growth, caries or injury. Internal lesions of the cord are generally painful; external lesions generally painless. Only the legs are paralysed; sensation of all kinds in both legs is generally disturbed.

Primary lateral sclerosis (generally wrongly diagnosed) is not common. It is usually due to syphilis. No sensory disturbance occurs. It first shows itself by causing weakness in the legs, followed by rigidity and paralysis. Sometimes the arms also become spastic. The abdominal reflexes early disappear. Increased knee-jerks, ankle-clonus and the other usual signs of spastic paraplegia are present.

Amyotrophic lateral sclerosis differs from most causes of spastic paralysis in that there is much wasting. The onset is rather insidious, and the hands and arms are affected as well as the legs.

The disease is progressive. There is no anæsthesia.

Syringo-myelia generally first shows itself in the hands, then in the arms, very slowly progressing to trunk, neck and legs. Deformities like Charcot's joint often arise. There is no ataxia. In some parts of the skin, heat cannot be told from cold, nor pain from touch, although ordinary sensibility remains.

Hæmorrhage into the cord is generally of traumatic origin.

Meningitis is generally recognised before paraplegia results.

Bilateral cerebral hæmorrhage or softening generally presents a history of two successive hemiplegic strokes. Arms and face are most likely to be affected, and the general history is likely to have diagnostic value.

FLACCID PARAPLEGIAS.

These result from lesions of the lower neuron, and are generally marked by flaccidity of the muscles, with wasting. The reflexes are lost or impaired, and the reaction of degeneration is present. Among children, the three principal causes of this form of paraplegia are the following :—

Acute anterior polio-myelitis usually begins as a slight febrile illness, lasting but a few days, and leaving certain groups of muscles weak or powerless, most frequently the extensor muscles of the toes and ankles. The onset is rapid, and there is generally an early improvement to a certain point, but usually some muscles are left permanently weak or paralysed. These are generally not affected bilaterally.

Tooth's peroneal type of progressive muscular atrophy is a familial

disease which first shows itself as a febrile illness, followed by inability to elevate the big toes. Inability to elevate the other toes and to flex the foot towards the shins follows. Later, the upper legs and the arms are affected.

Peripheral neuritis in children generally follows a recent attack of diphtheria. It often closely resembles anterior polio-myelitis. Paresis of the soft palate points to neuritis, but a history of recent diphtheria is the most helpful diagnostic differential.

Peripheral neuritis in adults is the commonest cause of flaccid paraplegia. It may be confused with acute anterior polio-myelitis, which occasionally starts in adult life. The onset of the latter, however, is more or less sudden, and partial recovery is rapid, usually leaving one or more groups of muscles affected. In peripheral neuritis there is usually pain or

tingling in the parts affected. In *alcoholic neuritis*, the extensor muscles of the legs are generally first affected. In *lead poisoning*, it is generally the extensor muscles of the wrists and the hand muscles that first show signs.

Care should also be taken to exclude *pelvic tumour*, pressing on the lumbo-sacral plexus ; and also *tumours interfering with the cauda equina.*

Transverse myelitis, at the level of the lumbar enlargement of the cord, also may produce symptoms at first not unlike those of peripheral neuritis.

PARAPLEGIAS NOT STRICTLY OF SPASTIC OR FLACCID TYPE.

In children, paraplegia may result from *primary muscular lesions.* In these cases, any reaction or reflex is of the normal type.

Pseudo - hypertrophic muscular paralysis is characterised by pronounced and increasing weakness of the legs, unaccompanied by apparent wasting of muscles or by any abnormal nerve reaction. The muscles of the hands and feet are rarely affected, but gradually most of the muscles of the body are involved, some wasting from the first, others apparently first becoming hypertrophied and afterwards wasting. Sensory disturbances are absent. The child cannot stand on tiptoe, and waddles when he walks. In order to rise from a lying position, the child is said to "climb up himself."

Primary muscular dystrophy closely resembles the disease just described, but wasting occurs from the first without any preliminary hypertrophy.

Among adults, the chief remaining causes of paraplegia are *tabes dorsalis*, *general paralysis of the insane*, *and functional paraplegia.*

Landry's paralysis generally occurs in early manhood. Its onset is rapid, a short period of malaise being followed by weakness in one or both legs. Paralysis of the legs is quickly established, and the muscles of the external trunk and diaphragm follow. There is no disturbance of sensation. It often disappears or ends fatally in a few days.

Tabes dorsalis starts insidiously and develops slowly. Ataxy of the spinal type (see page 33) soon begins to manifest itself. Tendo-Achilles jerk generally disappears first, then the knee-jerk. No

R.D. Argyle Robertson pupils (reaction to accommodation, but not to light). No wasting. Straight line test and Romberg's test (see page 33) both show ataxy. Gastric crises, lightning pains, Charcot's joints and perforating ulcer of the foot may show themselves even before the Argyle Robertson pupil. Optic atrophy is sometimes the first symptom of the disease.

G.P.I. does not produce paraplegia until a late stage of the disease, when its recognition will have been determined.

Functional paraplegia presents no wasting, no alteration of plantar reflexes, no ankle-clonus, and no R.D. Anæsthesia, if present, is not distributed in accordance with the distribution of any particular nerve or nerves.

ATAXY.

Power to co-ordinate the movements may best be tested by directing the patient to walk along a marked straight line, placing one foot directly in front of the other; to button and unbutton his clothes; to place each of his forefingers in turn on the tip of his nose; and to stand with his feet as near together as will enable him to stand steadily with his eyes open, and then to close his eyes (Romberg's sign) and, with his eyes closed, to move each of his arms and legs in turn to a position similar to that to which its fellow has been moved by the examiner.

When the ataxy is due to a peripheral nerve lesion, the symptoms are usually bilateral; the extensor areas are usually most affected; there is generally diminished cutaneous sensibility to pinpricks of hands and feet, but increased painful sensibility to

deep muscular pressure in those parts.

The ataxy is more pronounced if the eyes are closed.

When the ataxy is due to a lesion of the spinal cord there is diminished sensibility both to pin-pricks and to deep muscular pressure of the limbs. Romberg's sign is usually observable, and there is difficulty in carrying out the straight line test, especially if the eyes be closed.

Cerebral ataxy is usually one-sided, or hemiplegic. It is increased if the eyes are shut.

Cerebellar ataxy may be unilateral, or bilateral. It is not materially increased if the eyes are shut. Giddiness is usual. The gait is not unlike that of drunkenness. There is generally nystagmus.

TREMORS.

The fine tremors associated with *muscular fatigue*, *weakness*, *nervousness*, *emotion* and *cold* are rarely difficult to diagnose. The tremor of *old age* affects mostly the head and arms. It is bilateral, and ceases when at rest. The characteristic signs of paralysis agitans, such as rigidity and muscular weakness, are absent.

Paralysis agitans rarely shows itself before middle age. It generally begins in one hand, and gradually spreads to the other hand, feet and head. The tremor is usually lessened during voluntary movement, and persists during rest, sometimes even during sleep. The fingers often look as though engaged in making bread-pills. Later, the feet may chatter when placed on the ground. The gait, in a pronounced case, is a sort of running, hesitating shuffle. There is a tendency, in walking, to start

slowly and then to hurry forward as if to save falling. There is increasing weakness and rigidity of the muscles affected. The speech is usually slow and high-pitched. There is a set facial expression. The deep reflexes are normal or increased. The mind is unaffected.

General paralysis of the insane. The mental changes generally show the nature of the case. The tremors are usually irregular in rhythm and extent.

Graves' disease often causes a fine, rapid tremor of the hands, and sometimes of the arms, legs, and even the whole body, on exertion. The other signs of this disease will easily serve to distinguish it.

Lead poisoning sometimes produces a fine tremor of the limbs or lips. Signs of plumbism will be found.

Alcoholism may lead to a fine tremor, most marked in the hands

and feet. It is increased on voluntary movement, and disappears during rest. The facial muscles often exhibit a curious, rapid, irregular twitching.

Chronic hemiplegia, and certain other cerebral conditions, often cause clonic contractions of great rapidity, and should be borne in mind.

Disseminated sclerosis affords the most striking instance of intention tremor—that is, tremor only accompanying, or greatly increased during, voluntary movements. The arms are most strikingly affected in this way. The oscillations increase in ratio to the degree of precision needed and aimed at. The characteristic signs of this disease are epitomised on page 23.

Various ataxias, such as cerebellar ataxy and Friedreich's ataxy, often produce an intention tremor; but the ataxia should help to show the nature of the disease.

COMA.

Unilateral paresis or paralysis (indicated by unequal pupils, unequal puffing out of cheeks, unequal limpness of arms or legs, or inequalities of knee-jerks or plantar reflexes) indicates brain lesion. Injury of the scalp or skull may be associated with the cause of the coma, or with the fall consequent on it. Cerebro-spinal fluid or blood escaping from the ears or nose points to the likelihood of *fractured base of skull.*

Cerebral hæmorrhage, apart from physical injury, usually occurs after middle age. The blood pressure is generally high; the heart often enlarged. The coma is not generally abrupt in its onset. Cerebral hæmorrhage—especially pontine hæmorrhage—may not produce unilateral paralysis.

Pontine hæmorrhage causes a high body temperature and contracted pupils.

Cerebral embolism is commoner in young people than in old. Organic heart disease is nearly always present. The coma is generally abrupt in its onset.

Opium poisoning causes contracted pupils and subnormal body temperature. The coma gradually deepens.

Carbon monoxide can generally be detected by the smell of gas, and by the pink colour of the patient.

The history of the case and the examination of the urine for sugar, albumen and lead will assist in the diagnosis of *diabetes*, *uræmia*, and *Saturnine encephalopathy*. Remember that albumen sometimes results from renal congestion following cerebral hæmorrhage.

Alcohol, *cold* and *heat stroke* should also be borne in mind as possible causes of coma. *Nervous shock* and *hysteria* may also be

thought of when all other causes seem excluded.

Epileptic coma may have been preceded by a convulsion in which the tongue has been bitten or urine passed. Also a history of previous fits may be obtained. Reflex sensibility persists.

It should be remembered that coma sometimes occurs in the course of *disseminated sclerosis* and of *general paralysis of the insane.*

A FEW EYE MEMORANDA.

Acute Inflammation.—It is important early to recognise iritis and glaucoma, both of which are sometimes confused with conjunctivitis. In *iritis*, the ciliary vessels are injected and dull red, the congestion being most noticeable at the corneo-sclerotic margin, and not disappearing on pressure; whereas in conjunctivitis it is the conjunctival vessels which are injected bright red, the congestion being most noticeable away from the corneo-sclerotic margin towards the circumference, and lessening on pressure. In iritis, the conjunctival lining of the lids is not inflamed. In *glaucoma*, both sets of vessels, conjunctival and ciliary, are congested, and the cornea is hazy and insensitive (whereas it generally remains clear and sensitive in iritis and conjunctivitis). The iris is injected in

glaucoma and iritis, but retains its colour in conjunctivitis. The pupil is active and black in conjunctivitis, but inactive or fixed in iritis and glaucoma, being small in iritis and dilated in glaucoma. In iritis, the iris is apt to become adherent to the lens at various points round the edge of the pupil, which thus looks irregular in outline when dilated by atropine. Glaucoma generally occurs after middle age, and is quickly marked by increased tension, pain, and rapidly failing vision. The eye feels hard to the touch. Remember that atropine is essential in iritis, fatal in glaucoma.

Failing Vision.—It is important early to recognise *progressive optic atrophy* and *toxic amblyopia*. The latter is distinguished by gradually deteriorating vision—equally in both eyes; the field of vision not being contracted. There is generally a small blind central

area, most marked for colours—especially green and red—but also for form. To test for the colour scotoma, hold a half-inch-wide strip successively of red and of green cardboard, so that half an inch shows above the hand holding it, about fourteen inches from the patient's eye (the other eye being covered), in the direct line of vision. The patient is instructed still to look straight ahead, but the coloured strip is moved two inches to the nasal side of the direct line of vision. In the first position, if a scotoma exists, the colour is not recognised, whilst in the second position it becomes obvious. The test needs to be applied more than once. In progressive optic atrophy, this colour-blindness is not confined to a central area. Changes in the fundus present themselves at an early stage, patches of grey discolouration soon showing in the disc.

DEAFNESS.

Rinné's Test.—Strike lightly a tuning-fork which vibrates 256 times a second, furnished with a flat foot-piece. Hold the foot-piece against the mastoid process of one side. Immediately the patient indicates that he no longer hears the sound hold the tuning-fork close to his ear without touching it. If, with a defective ear, the sound is then heard again (positive Rinné), labyrinthine deafness is indicated; if it is not heard (negative Rinné), some trouble in the outer or middle ear is probable. In a normal ear, a positive Rinné is given.

Weber's Test.—This is of value when deafness is one-sided. Hold the foot-piece of the vibrating tuning-fork against the middle of the patient's forehead. If the patient hears the sound best on the deaf side, the trouble is in the

outer or middle ear, or the Eustachian tube is occluded. If he hears it best on the good side, the trouble is labyrinthine. If the trouble is in the conducting apparatus, examine for wax, foreign bodies, and polypi in the outer ear ; as well as for inflammation of the middle ear, oto-sclerosis, catarrh of the Eustachian tube, or its obstruction by enlarged tonsils or adenoids (especially in children)

GENERAL ABDOMINAL DISTENSION.

Ascites.—Abdominal distension is uniform bulging at most dependent part, which varies with position of patient. The umbilicus tends to become stretched flush with the general abdominal surface. The greatest abdominal circumference is generally at umbilicus, which is usually nearer to the pubes than to the ensiform cartilage. Dullness on percussion is marked at the most dependent parts, the highest parts being resonant, thus varying with position of patient. With the patient lying on his back, a thrill can be felt by the hand placed flat against the flank if a sudden pressure be applied to the other flank by the fingers of the other hand. The fluid drawn off by paracentesis is usually yellow, thin and fairly clear. If ascites follows œdema of the legs, it is generally due to chronic failure of the right heart

—either muscular or valvular—or to obstruction of the inferior vena cava through thrombosis of mediastinal tumour. If general œdema accompanies, but does not precede or follow, ascites, *Bright's disease* is usually the cause. If there is no other fluid swelling. or if such occurs long after the ascites manifests itself, usually the cause is *tubercular* or *malignant peritonitis*, or *obstruction of the main portal vein* by a thrombus, or by pressure of enlarged lymphatic glands—tubercular, malignant, etc.—or by pressure caused by adjacent tumour or by cirrhosis of the liver. If the ascites is accompanied with jaundice, the diagnosis is usually between *cirrhosis* and *malignant disease*. The history of the case, and the state of the liver and other organs, generally serve to distinguish between the two.

Phantom Tumour. Swelling disappears under anæsthetic.

Distended Bladder. Swelling central. On percussion, centre dull, flanks resonant. Incontinence usual. The passing of catheter removes swelling.

Ovarian Cyst. Swelling at first unilateral, and umbilicus usually at first nearer to one anterior superior iliac spine than to the other. Umbilicus usually nearer to ensiform cartilage than to pubes. On percussion, usually dullness in front, resonance at flanks. Greatest abdominal circumference usually below umbilicus. The fluid drawn off by paracentesis is usually dirty brown or greenish, thick and cloudy.

Tympanites. No fluid thrill. On percussion, resonance both in flanks and at front, in all positions.

The possibilities of *obesity* and of *pregnancy* should be borne in mind.

JAUNDICE.

Jaundice which clears up in a few days or weeks without much pain is probably *catarrhal*, especially in young people. Jaundice following severe colic is probably due to *gall stones*. Recurrent attacks of jaundice in middle-aged women, with long intervals of months or years, are generally due to *gall stones*. (Remember that gall stones do not negative malignant disease.)

In *chronic pancreatitis*—which closely resembles this in signs—the jaundice lasts longer, and rarely quite disappears. Persistent jaundice, with enlarged gall-bladder, in middle age, generally indicates *carcinoma of pancreas*. Pancreatic disease is usually accompanied with deep-seated, epigastric pain, and with fat in the stools. Pancreatitis and cancer of the pancreas usually cause a positive reaction to Cammidge's test. (This test

consists in hydrolysing the urine, having removed any sugar by fermentation, by boiling it with dilute hydrochloric acid, neutralising the excess of acid with lead carbonate, adding iri-basic lead acetate, and then treating with phenylhydrazine. A positive reaction consists in the formation of a golden deposit of hairlike crystals arranged in sheaves, easily soluble in dilute sulphuric acid.) Too much reliance, however, must not be placed in this test, unless clinical symptoms are also taken into account. Persistent and increasing jaundice with wasting and without much pain, in middle age, is nearly always *malignant* in causation. Always examine for rectal cancer.

In *Cirrhosis*, the edge of the enlarged liver is even and without nodules. The history is important. The surface may be hobnailed. Generally much ascites. Pyrexia may occur with jaundice, whatever

its cause; but the absence of pyrexia negatives *typhoid*, *Weil's disease*, *hepatic abscess*, *cholangitis* and *pylephlebitis*.

Syphilis of the liver often causes intense jaundice. Ascites may be present or absent. The history and other evidence of syphilis may throw light on the nature of the case.

A FEW URINE POINTS.

Sugar in Urine.—Fehling's test is usually the most satisfactory; but, if the urine is strongly alkaline, it should be made slightly acid by adding acetic acid. Mix in a test tube equal quantities of the two Fehling solutions, and boil. In a separate tube boil a little of the urine. Add a few drops to the heated mixed test solution, and boil again. A red deposit indicates glucose.

Legal's Test for Acetone.—Pour into a test tube 5 c.c. of urine. Add about ten drops of liquor sodæ, then a similar amount of fresh nitro-prusside solution, and lastly a few drops of strong acetic acid. If acetone is present, the reddish-brown colour produced by the nitro-prusside solution is deepened on the addition of the acetic acid. If acetone is absent, the colour disappears when the acid is added. When glycosuria has been

detected, the test for acetone should be applied at frequent intervals. Its presence with sugar indicates a much graver stage than does the presence of sugar alone. Improvement may best be gauged by the diminution of acetone. Acetonuria without glycosuria often occurs whenever there is carbohydrate lack, as in gastric ulcer, persistent vomiting, etc.

Hæmaturia.—When the bladder or lower urinary tract is the seat of the hæmorrhage, the urine is coloured bright red, unless the blood has been retained in the bladder for some time, when it may be dark-coloured. When the first urine passed is stained red, the remainder being clear, the hæmorrhage is probably prostatic or urethral. When the reverse occurs, the bladder is the probable source of the blood. Blood from the kidney generally produces smoky urine. If the

guiacum and ozonic ether test shows blood to be present, a microscopic examination of the deposit obtained by centrifuging the urine; a complete physical examination of the heart, lungs, liver, testes, etc.; a thorough rectal examination; a careful cystoscopic examination, and an examination by skiagraphy are all essential if an accurate diagnosis is to be obtained.

The guiacum and ozonic ether test consists in adding two drops of tincture of guiacum to about one inch of urine in a test tube, and then carefully adding, without shaking, about an inch of ozonic ether. If blood is present, a blue colouration appears where the fluids meet. This colour is not diffused throughout the mixture, as happens when iodides are present.

Acetic Acid Test for Albumen.—Pour into a test tube urine to occupy three-fourths of

the tube. Hold the bottom end of the tube, and heat the top layer of urine (by holding the tube obliquely over the flame) to boiling point. Cloudiness may be due to phosphates or to albumen. Then add 5 or 10 drops of B.P. acetic acid solution. If the cloudiness disappears entirely, it was due to phosphates. If effervescence occurs, carbonates were also present. If the cloudiness persists, it is due to albumen. It is well to make sure that the permanent cloudiness is not due to a deposit of nucleo-proteid brought down by the acetic acid itself. If a drop of non-fuming dilute nitric acid be added, the nucleo-protein cloud will disappear. An albumen deposit, if present, remains.

If medical practitioners will send us the names and addresses of professional friends to whom they think "The Doctor's Pocket Remembrancer" would be of interest, we shall be pleased to forward them copies.

Only by Trial and Experiment can a just opinion of the quality of ENO'S "Fruit Salt" be formed.

ENO'S "Fruit Salt" is a pleasant, effervescent, saline laxative, consisting essentially of combinations of alkalies with fruit acids. Salts such as sulphate of soda and the magnesium compounds are excluded. The best obtainable raw materials alone are used, and the product is analysed by our chemists at every stage of manufacture. Its agreeable taste owes nothing to artificial sweetening or flavouring agents. The utmost care is taken to ensure complete and uniform solubility; and this takes place without any stirring or shaking. It is claimed that in ENO'S "Fruit Salt" we have a palatable and thoroughly efficient agent for the removal of excessive intestinal bacteria, and of those protein decomposition products to which so much illness—minor and major—is ultimately due.

A sample Bottle of ENO'S "Fruit Salt" will be sent free to any Medical man upon application to J. C. ENO Ltd., 160 Piccadilly, London, W. 1.

Printed in Gt. Britain
by
ROBERT MACLEHOSE & CO. LTD.,
University Press, Glasgow
and published by
J. C. ENO LTD.
160 Piccadilly
London, W. 1