surgery in this patient was performed six days after wounding, and at operation the wound was foul-smelling and semi-purulent, and contained a piece of wood two inches long firmly plugging the skull defect and penetrating deeply into the brain. Many bone fragments lay deep in the brain substance; two of them were still visible in post-operative radiographs, indicating that wound excision had been imperfect.

The non-occurrence of a cross-infective discharge from these 40 wounds (except in one case of superficial infection) clearly suggests that when such discharge occurs it is more probably due to hospital than to initial infection.

It has been shown by Cowan (1938) that organisms occurring in the air and normally regarded as saprophytes may be the cause of serious intracranial infection, and this was later emphasized by Cairns (1939) in reporting a series of 23 deaths from infection following intracranial operations. In Cairns's cases the various organisms isolated had previously been considered non-pathogenic to man and were thought to have gained access to the cerebrospinal fluid spaces from the air during the prolonged exposure of the brain necessitated by some neurosurgical procedures. The effect of introducing *Ps. pyocyanea*, or any other pathogen, into the brain or cerebrospinal fluid spaces can well be imagined, and would be a matter for serious concern. If, therefore, penicillin is to be given by the method of local instillation the precaution of using an aseptic cap for the tube should certainly be taken.

No reflection upon the nursing staff concerned in the treatment of the second series of cases is intended: they have shown a capacity for hard work and co-operation which is admirable, and they could not have been expected to adopt and maintain a specialized system of ward asepsis and anti-sepsis which has been gradually developed over a period of four years. Nor could so small a number of nurses, even if trained and experienced in neurosurgical work, hope to attain a high standard (i.e., a low potential rate of hospital infection) when confronted with large numbers of patients with serious head injuries, whose sole ambition appeared to be the immediate removal of their headress.

Conclusion

We feel that attention to three main points is responsible for the very low rate of hospital or cross-infection of wounds in the above series. These points are: (1) The care taken in closing scalp wounds to obtain very early healing. (2) The insistence upon very careful ward and dressing technique as advocated in an earlier paper and the M.R.C. Memoranda on hospital infection. (3) The aseptic cap used to close the penicillin tube.

Summary

Two series of cases are reported, each of 20 battle casualties, in which local penicillin therapy was employed.

The incidence of potential cross-infection was three cases in 20 in the unit's original surroundings, and no clinical cross-infection occurred. In the second series, under less perfect conditions, the potential rate rose to seven cases in 20 (35%), and one superficial case of clinical hospital infection resulted (5%).

Cross-infection of wounds by way of the penicillin tube is an obvious possibility and, as it has not appeared in the present series (the one case described being only a superficial cutaneous infection), it is suggested that the aseptic screw cap in use is the responsible factor in prevention.

The need for proper closure of the scalp wound and very early skin-healing is emphasized in view of the total average potential hospital- (or cross-) infection rate (25%).

Our thanks are due to Dr. Lever and Dr. Cross, who carried out the necessary bacteriological investigation of the large number of wound swabs taken during the period under review, and whose admirable co-operation could not have been bettered.

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SOME MISTAKEN DIAGNOSES IN THE COMMON INFECTIOUS FEVERS

BY

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Recently I had occasion to go through the records of this hospital, and it occurred to me that it might be of general interest to sift carefully all those cases of "everyday" infectious disease in which my own diagnosis differed from that of the certifying practitioner. I have deleted all the doubtful cases, where almost anybody might be right: those to which I allude have been proved, so far as was possible, by bacteriological and pathological aids, for which I am indebted to Dr. Wordley of Plymouth, who is ever helpful in the elucidation of obscure medical problems.

Most senior practitioners will be well acquainted with the contents of this paper, but perhaps those who have had little opportunity for the study of infectious disease may derive some benefit. Many of us after qualification find the diagnosis of the infectious fevers difficult, and I am not sure that it becomes any easier the more cases one sees, with the consequent larger number of possibilities that arise with each fresh problem.

This brief review covers the last five years' admissions to the City Isolation Hospital, Plymouth, and includes 3,516 cases of diphtheria, 2,102 cases of scarlet fever, and a miscellany of 700 other diseases. I have tried to stress the clinical aspect throughout in view of the fact that pathological aid may well be wanting when needed most; but, naturally, in many of these cases the only ultimate proof lay with the laboratory, so it was essential to send these patients to a hospital where pathological facilities existed for full investigation. Nor do I wish to convey the impression that I am in any way critical of the differences mentioned, for I well know the heavy responsibility that rests with a practitioner who, perhaps living at a distance, is called to see some of these cases and is expected to give an infallible diagnosis in impossible surroundings within a few minutes. Quite rightly, he removes the case to where observation and treatment can be carried out under more favourable conditions. Still, there is always some satisfaction in making a correct mental diagnosis, irrespective of the administrative aspects of the disease.

Diphtheria

Diphtheria is the commonest infectious disease sent to the fever hospital in this district. I propose to divide the cases, 3,516 in all, into two groups—the faucial, nasopharyngeal, and nasal varieties, accounting for some 3,363 cases, and laryngeal, making up the remainder, a total of 153.

As can well be imagined, the commonest, safest, and most justifiable error in the first group is a diagnosis of tonsillitis. This occurs in 347 cases, or 72% of the total 483 mistaken diagnoses. Next comes scarlet fever, accounting for 14% (67 cases); and third on the list are various forms of Vincent's angina and stomatitis. The remainder is made up of the following diseases: Anginous form of glandular fever (4 cases), streptococcal cervical adenitis (4), post-tonsillectomy sloughs (2), chicken-pox (1), purpura (1), secondary syphilis (1), alveolar abscess (1), mumps (1), thrush (1), acute lymphatic leukaemia (1), and, quite inexplicably, a toxic erythema, a tuberculous meningitis, a case of meningococcal meningitis, and an attempted suicide; but to this last case I shall allude later.

The clinical differentiation of acute tonsillitis from an early diphtheria may be extremely puzzling at the outset, and I have noticed during the last few years that it is increasingly common for diphtheria to start just like an early follicular tonsillitis, and at this stage it is almost impossible without bacteriological aid to make a sure diagnosis. The following points may be borne in mind when faced with such a case:

1. In diphtheria of this type the onset is insidious. I have had nurses with a vague uneasiness in their throats report to me with considerable follicular exudate on one or both tonsils who only thought that something might be wrong when they looked at their own throats. The constitutional disturbance, pain on swallowing,

tender glands, and pyrexia are all minimal at this stage. Frequently only one tonsil is affected, but within 12 hours the other shows signs of involvement, and within 24 hours the exudate has coalesced, leaving no doubt as to the true nature of the illness.

2. Coalescence is quite rapid in diphtheria—within 24 hours. A discrete exudate lasting two or three days is unlikely to be diphtheria. It follows, then, that a case which at first sight appears to be tonsillitis should always be inspected again on the following day, and the day after that if possible.

In *streptococcal sore throat* the onset is more sudden, the systemic disturbance more severe, and the temperature higher than in diphtheria. The cervical glands are very tender, the surface of the exudate is rough, pultaceous in consistency, and may be associated with intense congestion of the soft palate and adjacent tissues.

Quinsy is most likely to be confused with hypertoxic diphtheria, as in both diseases there is exudate on the tonsils and oedema of the fauces. Again, in diphtheria of similar severity there would be much greater glandular enlargement—probably even a bull neck—pallor, and nasal discharge. Quinsy is mostly unilateral and is often associated with great difficulty in opening the jaws, a most uncommon sign even in the severest types of diphtheria. Very occasionally diphtheria does occur with quinsy in the acute stage, but almost always it is a sequel after the throat has cleared of membrane, presumably due to the lessened resistance of the patient, both locally and generally, to secondary invading organisms.

Other kinds of sore throat are encountered occasionally, but the ones mentioned are those which commonly give rise to difficulty.

There seems to be an impression that the temperature in diphtheria is always normal or subnormal. This is by no means true, and in fact most cases show mild pyrexia of 100–101° during the first few days of the illness, particularly in children. The presence of fetor, on which again much reliance is placed, can be misleading, and septic sore throats or mixed infections with Vincent's organisms can sometimes produce an odour which to the inexperienced is a very good imitation of the diphtheritic smell.

Scarlet fever is the disease next most commonly confused with diphtheria; many of the cases are overlooked by omitting examination of the patient's skin, and so the rash is missed. Sometimes the rash may have disappeared by the time the doctor is called in, but a retrospective diagnosis is usually possible by consideration of the history of sudden onset with vomiting and sore throat, and the characteristic appearance of the throat, tongue, and skin. Sometimes undoubted scarlet fever occurs without a rash, but again the history will usually provide a clue. Occasionally both diseases coexist.

Vincent's angina has been especially prevalent during the last two or three years, but even before the war it accounted for some 4% of the errors of diagnosis. It occurs not at all uncommonly in children, but adults are the chief sufferers. The disease mostly affects one tonsil and is readily recognized clinically by the ulceration, the broken and crumbly appearance of the exudate, the typical smell, and the tender glands, which are often enlarged out of all proportion to the extent of the angina. It should be noted that diphtheria is often associated with Vincent's angina, and swabs as well as smears should be taken to confirm the diagnosis. Vincent's disease will also attack the gums and buccal mucosa, causing much swelling of the gingival margins, ulceration of the buccal mucous membrane, and the secretion of tenacious mucus, a condition I have never observed in diphtheria.

The differential diagnosis of the remainder of the diseases mentioned above—namely, secondary syphilis, acute leukaemia, purpura, post-tonsillectomy sloughs, and non-diphtheritic rhinitis—are all fairly obvious, but there are three to which I would like to allude briefly: glandular fever, chicken-pox, and the attempted suicide.

The anginous form of glandular fever may simulate diphtheria very closely in children. There is considerable exudate on the tonsils, with faucial oedema and enlarged glands in the neck. A striking difference is to be found in the consistency of the cervical glands on gentle palpation. Instead of being enlarged and firm, they have a peculiar elastic and resilient feeling, quite

unlike those of diphtheria. Once one's suspicions have been aroused by this sign, further examination will in all probability reveal an enlarged spleen, adenopathy of other groups of lymph glands, and possibly some abdominal tenderness. A differential count will confirm the diagnosis, but, if a Paul-Bunnell test is done, blood should be taken before giving serum in doubtful cases, otherwise the result will be positive when in fact the true result might have been negative.

I have notes of several cases of chicken-pox misjudged as diphtheria. This has been due to a profuse crop of vesicles on the palate or uvula breaking down into coalescent ulcers. Again an examination of the skin would have settled the diagnosis.

Lastly, the attempted suicide, which is related merely as a matter of medical interest.

A man aged 45 was sent into this hospital certified as suffering from diphtheria. He had a curious-looking throat covered with dirty grey exudate, extending over the tonsils, faucial pillars, uvula, and soft palate. The mucous membrane of the cheek was involved, and it was quite easy to strip off this material, when a raw, sore area was left behind. I had never seen anything quite like it before. His general condition seemed good. He was not ill, but was rather confused in his manner. On further inspection I found a severe wrist wound on the left side which had evidently been self-inflicted by a sharp instrument—probably a safety-razor blade. The flexor longus pollicis had been divided but no large blood vessel injured. I taxed the man with attempted suicide, as by this time it was evident that he had swallowed some corrosive fluid and, perhaps finding this rather unpleasant, attempted a short cut, which was equally unsuccessful. He would admit nothing, but later I learned that he was heavily in debt and had been found wandering on several occasions.

Perhaps, however, laryngeal diphtheria is confused more than all the other kinds put together, with the possible exception of skin diphtheria. Out of 153 cases notified 38% proved not to be laryngeal diphtheria after investigation and observation. These difficulties are quite understandable, as the number of cases in the large fever hospitals has fallen very considerably in recent years and not many of the younger practitioners have had the opportunity of seeing these cases before qualification. In addition, a doctor called to see suspected cases of "croup" quite rightly errs on the safe side, so that a number of patients arrive at the hospital with this label who have no signs of the disease.

A bad faucial diphtheria with palatal oedema and noisy breathing due to mechanical causes in the throat will often be diagnosed as laryngeal obstruction, but the chief mistakes from the academic point of view arise in the confusion between the various forms of catarrhal laryngitis, the laryngitis present at the onset of an attack of measles, bronchopneumonia, and retropharyngeal abscess. Other occasional difficulties arise, and I have notes of odd cases of papilloma of the larynx, congenital laryngeal stridor, and whooping-cough.

Catarrhal laryngitis, or spasmodic croup, accounts for 55% of the total number of errors, and this is quite feasible when one thinks of the alarming picture presented by a child with this complaint. The chief clinical contrasts between this and laryngeal diphtheria are:

1. Its sudden onset, very often at night, in an otherwise healthy child, whose only previous history may have been that of a slight upper respiratory catarrh.
2. The attack reaches the acme of severity almost at once and the child is fairly well between each spasm. Often this type of patient is quite well by the morning.
3. The laryngeal involvement is never progressive, although occasionally it may last some days.

Compare this picture with that of a true diphtheritic laryngitis, which is insidious in its onset. Perhaps there may be a history of sore throat, in which case the presence of exudate on the tonsils will make the diagnosis easy. Often, however, the parents may have been unaware of the true state of affairs, and only when the child loses its voice, breathes noisily, or has fits of dyspnoea are their fears aroused. A large number of these cases have clean throats when examined, as the membrane has separated from this situation by the time the larynx is involved. As the attack has probably been severe and of the nasopharyngeal variety, much useful information may be gained by looking carefully at the throat for evidence of slight super-

ficial ulceration, suggesting recently separated membrane, and also the nose for evidence of nasal discharge or actual membrane, which often persists after the throat is clean. In addition, late cases also have a greyish pallor and may still show signs of subsiding cervical adenitis. The characteristic feature of all diphtheritic laryngitis is progression in the absence of treatment. Sometimes it is slow, sometimes rapid, but most cases will terminate fatally unless the true nature is recognized. Unlike other forms of laryngitis, where a child may be almost normal between attacks, in diphtheria the stridor and recession get steadily worse, restlessness increases, and the attacks of dyspnoea are more frequent. Primary laryngeal diphtheria is less common, but accounts for some 20% of those requiring tracheotomy.

Measles.—The next most frequent disease (17%) sent into hospital as laryngeal diphtheria is measles. Not uncommonly, before the appearance of the rash the child may show all the signs of impending laryngeal obstruction, and unless Koplik's spots are identified the diagnosis will be in doubt. As a rough guide, laryngeal symptoms occurring before the rash are due to measles. Laryngeal symptoms appearing after the rash are probably diphtheritic. Laryngeal involvement with the rash may be either, for diphtheria is more often associated with measles than with any other infectious disease, with the exception perhaps of scarlet fever. The presence of laryngeal obstruction associated with measles, whether due to diphtheria or not, formerly had some prognostic significance, for if tracheotomy was found necessary the outcome was nearly always fatal, but I have recently recorded four successful cases whose recovery was probably due to sulphapyridine.

Retropharyngeal abscess should never be confused with true laryngeal diphtheria. The peculiar quacking cry, the noisy breathing, and the presence of a fluctuating swelling on the posterior pharyngeal wall, which can be seen and felt, and of enlarged glands, usually on one side of the neck, all point to the correct diagnosis. Pneumonia, bronchitis, and whooping-cough are all misdiagnoses which can be avoided by careful assessment of the patient's history, signs, and symptoms.

In all these conditions diagnosis on clinical grounds is important, and the laboratory should be used for confirmatory evidence only after the necessary and immediate specific treatment has been given.

Considerable assistance in the elucidation of a doubtful case may be obtained from a knowledge of the patient's immunity in response to a Schick test and the typing of the organism. If the case is obviously doubtful, and there is no immediate danger in withholding serum for a few hours, the patient should be Schick-tested and swabs taken from the nose and throat. Serum may then be given in 7 to 8 hours without upsetting the result of the Schick test. The following results may occur, bearing in mind that all intermedius and gravis strains of diphtheria bacilli are virulent and that mitis strains may be either virulent or non-virulent.

Schick positive ; swab positive		Diphtheria
Schick positive ; swab negative		Susceptible, but probably
		not diphtheria
Schick negative ; swab positive		Immune ; ? carrier
Schick negative ; swab negative		Not diphtheria

All swabs should be repeated as a confirmatory measure. In any suspicious case, particularly in private practice, serum should be given first and confirmatory swabs taken subsequently. The technique just described is more suited to a hospital, where a close watch can be kept for possible extension of the disease and suitable treatment undertaken immediately.

Scarlet Fever

There is some doubt whether scarlet fever should be regarded as a distinct disease or merely as one variety of the response of the individual to the toxin of a particular strain of haemolytic streptococcus ; but, nevertheless, it is a notifiable disease which is usually segregated, and it is therefore necessary to arrive at a clinical diagnosis without considering too critically its exact mechanism.

Out of over 2,000 cases the diagnostic error is some 8%, composed of the following: toxic erythema (26% of errors), tonsillitis (20%), rubella (18%), measles (12%), diphtheria

(6%). The remaining diseases involved are scabies, urticaria, chicken-pox, impetigo, industrial dermatitis, and glandular fever. Perhaps the commonest sources of error are: (1) Taking one manifestation only of the disease, such as the rash, and diagnosing on this alone: scarlet fever is the sum-total of a number of signs and symptoms. (2) Paying too little regard to the history. (3) Omitting to look at the patient's skin, or arriving at an erroneous conclusion after inspection.

Toxic Erythema.—Thus the presence of a scarlatiniform rash does not of itself mean that the patient is suffering from scarlet fever. I have grouped all these cases under the rather loose heading of "toxic erythema," and this comprises rashes attributable to drugs, chiefly atropine, enemata, rubbing-oils, food-poisoning, wearing of rough clothing or blankets next the skin, fits of crying in children (a fruitful source of "rash" after throat examination), and a host of others for which no adequate cause could be found. In almost all the rash dominated the picture, and the other symptoms, such as vomiting and sore throat, were absent. Many of these patients subsequently desquamated, but the desquamation was unlike the pin-hole variety found in scarlet fever.

Tonsillitis accounted for the next 20% of the mistakes, and some of these were missed because no careful examination of the patient's skin had been made, the diagnosis being based on the appearance of the throat and tongue ; and some because a transient rash, not an uncommon event even in tonsillitis, had appeared and a wrong conclusion had been deduced. The rashes seen in tonsillitis are erythematous—not punctate—patchy in distribution, and of short duration.

Rubella.—Problems of real difficulty may, however, arise in distinguishing a second-day rubella from a mild scarlet fever, and I have come to the conclusion that it is well-nigh impossible in certain cases. Some 18% of the errors occurred in the differential diagnosis of these two diseases, and there were some doubtful cases which have not been included. I noticed, particularly in the last epidemic of rubella, that a number of undoubted cases had considerable faucial congestion, and when on the second day the rash had disappeared from the face, leaving a scarlatiniform erythema on the rest of the body, it was very difficult sometimes to make the necessary distinction. In addition to the usual distinguishing features of German measles—namely, mild generalized adenopathy, various muscular pains, catarrhal symptoms, and pyrexia—there is one physical sign which is very constant in patients with clean mouths, and this is a marginal gingivitis. On inspection of the mouth the gums are found to be red, swollen, "heaped up," and overlapping the teeth, particularly between the upper centrals and laterals. This sign in conjunction with the other well-known manifestations of the disease will often be found most helpful.

Measles and Varicella.—The two diseases which so often simulate scarlet fever and which account for another 14% of the pitfalls are measles and chicken-pox. Measles is nearly always confused with scarlet fever in the prodromal stages of the disease, one or two days before the true rash appears, when the body may be covered with an eruption indistinguishable from scarlet fever, and the throat and tongue may well be passable imitations of the latter disease. A close inspection of the buccal mucosa will reveal Koplik's spots and quarantine of the scarlet fever ward be avoided! The same remarks apply to chicken-pox, in which a scarlatiniform rash may appear before the true rash. An inspection of the palate will usually reveal one or two early vesicles, giving the key to the situation. To add to the difficulties here, it is not infrequent for scarlet fever and chicken-pox to occur at the same time ; but true scarlet fever usually appears after the chicken-pox is fully developed and shows the signs appropriate to that disease.

I have no space to deal with the less important diseases sent into hospital as scarlet fever, such as urticaria, scabies, and impetigo, and their differentiation is obvious enough. Clinical diagnosis is still the best means of recognizing scarlet fever. The Dick and Schultz-Charlton tests and results of throat swabs are often misleading and inconclusive, and I rarely employ them now. A helpful sign in scarlet fever which aided in making a retrospective diagnosis in a doubtful case was the

appearance in due course of pin-hole desquamation; but it is well to remember that if scarlet fever antitoxin has been given early in the disease, subsequent desquamation often fails to appear.

Typhoid Fever

Typhoid fever is a disease of such protean manifestations that it is hardly surprising to find a total error of 37% in the 63 cases notified. I have not included as an error those cases which were sent in as typhoid and subsequently turned out to be other diseases of the enteric group, as clinical diagnosis is well-nigh impossible except perhaps in large-scale epidemics. The chief difficulties seem to arise with (1) various chest diseases; (2) other intestinal diseases; and each of these groups accounts for some 30% of the total faulty diagnosis. The remainder are found in such other cases as tuberculous peritonitis, acute military tuberculosis, pectoral abscess, pityriasis rosea, malignant endocarditis, and infective hepatitis. One other interesting alternative occurred recently, and this merits further description owing to its general medical interest.

Four cases of lobar pneumonia, one of bronchopneumonia, and one of empyema were noted; none of these were very obscure in their signs and symptoms, and would most certainly have been excluded had a careful examination of the chest been made. As a rule, pneumonia as a complication occurs late in the disease, and the signs in the chest, coupled with the cough, dyspnoea, and pain in the side, are all against the diagnosis of typhoid fever except in the rare form of pneumo-typhoid in which the disease has all the symptoms and signs of lobar pneumonia at the outset. In this connexion it is perhaps worth while noting that herpes of the lips is very unusual in enteric fever. In fact, a critical examination of the patient's chest and a blood smear showing leucopenia and absence of eosinophils would automatically rule out pneumonia and many other conditions superficially like typhoid fever.

Generalized military tuberculosis may cause real difficulty, but the early loss of weight, rapid pulse, and possible leucocytosis may help to distinguish it. Jaundice is so rare in typhoid fever that its mere presence is sufficient to exclude the disease, and the only case in this series showing jaundice proved to be infective in origin. With regard to intestinal diseases the simple enteritis lacks the quiet and insidious onset of enteric. The sudden vomiting, gripping abdominal pain, diarrhoea, and tenesmus are all unlike the vague feeling of real illness which ushers in the onset of typhoid fever. Malignant endocarditis may simulate typhoid very closely: the high temperature, enlarged spleen, perhaps abdominal distension, all combine to make an exact assessment difficult. A search for a possible cardiac lesion, haematuria, and petechiae in the skin will help in excluding the former disease.

One case of special interest occurred quite recently at the hospital. A man 32 years of age, a baker by trade, was admitted with a diagnosis of typhoid fever. He had been ill at home for two weeks with severe headaches, insomnia, pains in his limbs, a high temperature, and gastro-intestinal upset, vomiting, and loose stools. On admission his temperature was 104°, pulse 132, respirations 36; and the only physical signs I could find were a slight icteric tinge in the conjunctiva, diminished air entry at the left base with a few post-tussive rales, an enlarged thyroid, and muscular pains and tenderness in the upper arms and calves of his legs. A white count revealed a leucocytosis of 20,000—polymorphs 62%, lymphocytes 18%, eosinophils 17%, and monocytes 3%. In view of his high eosinophil count I made careful inquiries as to his occupation and habits of life. It turned out that he was a pork-pie baker and that every day he ate a few ounces of raw pork to see if it was sufficiently seasoned before putting it in the pies. Suspicion was at once aroused as to the possibility of trichiniasis, and his subsequent signs and symptoms were very typical of the disease. The calf muscles became swollen and tender, his tongue was enlarged to such an extent that he could hardly protrude it, and the swelling of his thyroid remained. The muscles of both upper extremities were soon involved, and he was very ill. At no time was there any facial oedema. Gradually the high fever settled, the muscular tenderness and swelling abated, and his thyroid returned to normal size. At this stage I removed a piece of his left deltoid muscle, and on examination this was found to contain *Trichina spiralis*. He was eventually discharged quite fit and well. I mention this case in greater detail as a very uncommon possibility when confronted with a case of "P.U.O.," and also to draw attention to the fact that a man's occupation often involves processes not imagined by the interrogator.

Perhaps the most helpful points in considering a doubtful case of typhoid fever and before sending specimens to the laboratory are as follows:

1. Gradual onset with headache and malaise.
2. A relatively slow, perhaps dicrotic, pulse with a rising temperature.
3. Constipation or diarrhoea.
4. Low blood pressure.
5. Abdominal distension, with palpable spleen.
6. Bronchitis and epistaxis.
7. The state of the tongue and throat; the latter may be congested.
8. The rash when present, bearing in mind that it may be sparse and require a careful search.
9. Absence of abdominal reflexes.

Dr. J. D. Rolleston drew attention to this last sign many years ago, and, provided that other nervous diseases are excluded, I have found it to exist in a large number of proved cases.

Naturally, most cases will have to be confirmed either by blood culture or by agglutination tests, and newer technique in these laboratory methods enables differentiation to be made between an active infection occurring in an inoculated person and even the carrier state.

Acute Anterior Poliomyelitis

Acute anterior poliomyelitis appears to be an even more baffling disease from the diagnostic point of view, for out of a total of 30 notified cases there is an error of just over 50%, and these include a list of nervous diseases which would do credit to Queen Square Hospital, mechanical faults which could be seen in the out-patient department of any general hospital, and the usual makeweights which had apparently no connexion whatever with the disease. These nervous diseases were: cerebellar tumour, cerebral tumour, disseminated sclerosis, traumatic neuritis (drunkard's palsy), a transverse lesion of the spinal cord, a hemichorea, and a post-diphtheritic palsy, easily demonstrated by immobility of the palate, nasal voice, and regurgitation of fluids through the nose.

I feel quite incompetent to discuss differential diagnosis in nervous diseases, so will pass straight to the mechanical factors influencing locomotion which were sent in under this heading.

One boy, unable to walk, was found to be suffering from a septic sore on the heel, another from inguinal adenitis secondary to a sore on the foot, and yet another from a tuberculous hip; finally, we had a man with lumbago. A few cases of scarlet fever and influenza, and a pneumococcal meningitis, complete the list.

Perhaps a very brief summary of the features of the spinal type of the disease may help to distinguish it from some of the above-named conditions.

The pre-paralytic stage is associated with sudden onset, catarrhal symptoms such as coryza and sore throat, vomiting, pyrexia, and malaise—in fact, much like the onset of any acute infection. During this stage two signs are useful: the spinal sign, in which there is resistance to passive anterior flexion of the neck, and Amoss's sign, which is useful for older children. If the patient is sat up in bed and asked to fold the arms, he may attempt so to do, but quickly places his arms behind, so that the body weight is borne on a tripod, consisting of his arms, with the spine as the "third leg."

If these signs and symptoms are present it is quite justifiable to send the case into hospital with a provisional diagnosis of infantile paralysis. Often, however, it is impossible to recognize the disease in the pre-paralytic stage with any degree of certainty, although its presence in the district will often arouse suspicion at these rather suggestive symptoms. This stage may be succeeded by an evanescent meningeal stage in which headache, drowsiness, and irritability are prominent symptoms, or may proceed to the paralytic stage straightaway. It is often possible to overlook this if too much attention is paid to the constitutional signs and symptoms. I have seen this happen in hospital, and only a few weeks ago I missed a case which I was looking after personally.

Before the actual paralysis occurs there is much muscle tenderness, hyperaesthesia, and stiffness of the neck and spine, so that the patient greatly resents interference by the examiner. Rarely, all these prodromal signs and symptoms may be wanting, and the sudden onset of paralysis may be the first signs of illness. I have observed such a case in this hospital.

When the paralysis occurs it is usually maximal at the beginning, but I have notes of one case in which there was a four-day interval

between paralysis of the right shoulder-girdle and of the left leg, the latter occurring after the temperature had returned to normal—rather an unusual occurrence. The tendon reflexes are usually absent or greatly diminished in this phase and the paralysis is flaccid.

Many varieties of history, signs, and symptoms are seen in different individuals, and the encephalitic form may justifiably be regarded as a tuberculous or other type of meningitis, unless facilities exist for examination of the cerebrospinal fluid.

Cerebrospinal Fever

During the war most practitioners have had a fairly large experience of cerebrospinal fever, but nevertheless it is interesting to consider the diseases which at first sight were thought to be meningococcal meningitis. Some of these were indistinguishable from cerebrospinal fever on clinical grounds alone, and out of 140 cases notified there was an 18% error, much reduced if other types of meningitis are excluded. Sixteen such cases occurred—9 tuberculous meningitis, 4 benign lymphocytic meningitis, 1 pneumococcal, 1 streptococcal, and 1 syphilitic meningitis. Again some confusion has arisen in various chest conditions, mostly in children, presenting signs of meningism: two cases of lobar pneumonia, one of bronchopneumonia, and one of spontaneous pneumothorax were wrongly thought to be cerebrospinal fever, although the last-mentioned case presented very few signs or symptoms referable to meningitis. Among the miscellaneous conditions seen were transverse myelitis, aneurysm of the middle cerebral artery, and one case each of measles, rubella, and scarlet fever.

It is impossible to differentiate with certainty the various forms of meningitis clinically before submitting a specimen of the cerebrospinal fluid to the laboratory, but in all the cases which subsequently turned out to be of tuberculous origin there was a history of gradual onset, a vague prodromal period of ill-health, or a history of some chest trouble which gave a pointer to the possible origin of the trouble.

Pneumonia in children may simulate meningitis very closely. The sudden onset, the signs of meningeal irritation, the high temperature, and perhaps paucity of physical signs in the chest are all misleading.

The measles and scarlet fever cases were probably sent in through the mistaken view that the rash was that of meningococcal meningitis, but there were no other signs to suggest a meningeal lesion. The patient with rubella was sent in because she complained of a stiff neck. This particular symptom sometimes occurs several days before the rash appears, and may well give rise to some doubts when cases of the more serious disease are prevalent.

Miscellaneous

I have mentioned the commoner infectious diseases; there are others, and combinations of the various infections, which I could include, such as bronchitis, frequently sent in as whooping-cough, erysipelas, which has been confused with eczema, carbuncle, dacryocystitis, herpes zoster, and cavernous sinus thrombosis; but of all the mixture of odd infections the most frequent mistake is the case labelled "measles and bronchopneumonia." Perhaps, however, this mistake may be a matter of expediency rather than a lack of clinical acumen. Ordinarily, cases of uncomplicated measles are not admitted; but attach the magic word "bronchopneumonia" and a bed is found somewhere!

In spite of this there are a great number of honest errors of judgment, and I think the reason is probably that the doctor is called in either just as the rash is appearing or when it is just fully developed, so that the child is restless and looks ill. At this stage the fever is considerable—the temperature commonly elevated to 103–104°, there is considerable catarrh in the chest, the respirations are frequent and short, and there is nearly always a dry cough. The physical signs in the chest at this stage are those of a mild bronchitis, but the whole picture looks at first sight rather like a bronchopneumonia. Very soon, within 24 to 36 hours, the rash begins to fade, the symptoms abate, and the temperature drops, and it is during the next few days that true bronchopneumonia is likely to develop. It is only exceptionally that pneumonia occurs in the prodromal stage or coincidentally at the time of eruption.

I have tried, so far as is possible, to avoid figures in this paper, nor do I wish to imply any criticism of other doctors' work. I am sure there would be ample material for another paper based on my own personal mistakes. Anyhow, it will show that life in a fever hospital is not so dull, that it does not consist entirely in looking after cases of scarlet fever, and that good clinical work can still be found in this branch of medicine—despite the war and the forms!

I have to thank Dr. T. Peirson, medical officer of health, City of Plymouth, for permission to publish this paper. I should also like to acknowledge the help I have obtained from the standard works on infectious disease for reference on doubtful points, particularly *Harries and Mitman* and my old chief's book, *Rolleston and Ronaldson*. Few who have worked under Dr. J. D. Rolleston could fail to be stimulated by his magnificent teaching on the infectious fevers.

GASTRO-INTESTINAL BLEEDING DUE TO VITAMIN K DEFICIENCY COMPLICATING LABOUR

BY

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In 1929–33 a haemorrhagic chick disease was observed in a number of laboratories (Brinkhous, 1940), in most cases incidental to experiments in which chicks were fed purified diets low in fats. During observations on the effect of various proteins on the growth of chicks spontaneous haemorrhages were noticed. Administration of lemon juice, ascorbic acid, cod-liver oil, and wheat-germ oil failed to prevent these haemorrhages in chicks fed on the purified diets. It was suggested by Dam, in 1934, that this condition constituted a new dietary deficiency disease, and his suggestion that the deficient factor be called vitamin K has been adopted. These diets were low in fats, and McFarlane, Lane, and Richardson gave evidence of the fat-soluble nature of the vitamin: they noted that fish meal prevented haemorrhage unless it had been extracted by ether.

Haemorrhagic chick disease was recognized as being due to deficiency, and the relationship of this dietary factor to blood clotting was established. A low plasma prothrombin was suspected: a haemorrhagic tendency appeared when the low prothrombin levels were reached when feeding chicks on K-deficient diets for several days. Both the low prothrombin and the haemorrhagic tendency were cured by feeding vitamin K.

That mammals may develop such a deficiency was shown in conditions in which the absorption of this fat-soluble vitamin is hindered by absence of bile in the intestine. Hypo-thrombinaemia and haemorrhage ensued, and were cured by the administration of vitamin K. Vitamin K occurs richly in alfalfa, spinach, cabbage, cauliflower, carrot-tops, and soya beans; it is less potent in tomatoes and orange peel. There is a little in liver and egg yolk.

The causes of vitamin K deficiency are deficient diet, obstructive jaundice, and biliary fistula leading to absence of bile in the intestine. Haemorrhagic disease of the newborn shows a low prothrombin level and is controlled by vitamin K. Jaundice appears to have no relation to the deficiency. Haemorrhage due to low plasma prothrombin may occur in ulcerative colitis, intestinal obstruction, and fistulae.

Attention is directed to a most complete and excellent review of the literature on plasma prothrombin and its relationship to vitamin K by Brinkhous (1940).

In view of the above facts it may be interesting to record a recent case of mine treated with vitamin K.

Case Report

A farmer's wife was taken ill in Sept., 1943, with acute pain in the back and vomiting. Her temperature varied from 100° to 103° for four days. She was diffusely tender over the abdomen, but no definite localizing signs were obtainable. Her condition becoming worse on the evening of the fourth day, I sent her into hospital, twelve miles distant. The urine showed no abnormalities up to that time. In hospital her case was diagnosed as a simple catarrhal jaundice, and bile appeared in the urine. A blood count revealed