and whether diluted or undiluted. They concluded that the minimum interval between feeds must be three hours.

Here is a chart, taken from an article by Dr. Harold Waller, which illustrates the effect on milk production of too frequent feeding. The child was an inveterate screamer and was constantly being fed in the hope of achieving quiet. Note the poor weight curve and the small amount of milk taken, as shown by test feeds, up to the seventh week. During that week the mother was persuaded to limit the feeds to six in twenty-four hours and in a fortnight to five feeds; the child became quiet and contented, and you will note the immediate improvement in the weight curve and in the quantity of milk taken at a meal.

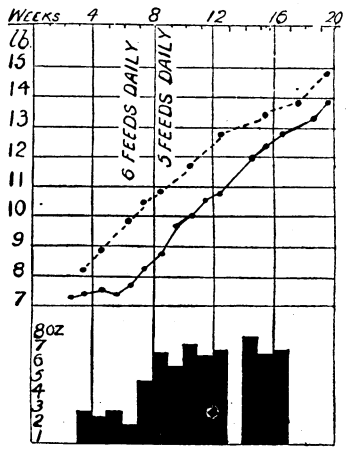

CHART 5.

Failures at the Third Week.

Why is it that so many failures occur early, say at about the third week? Constantly I am being told, "The milk went when I got up." The figures I have quoted point to one important cause, but there must be others. The woman—at any rate the working woman—gets up on the tenth day and resumes almost full household responsibilities and anxieties. This in itself is quite sufficient to diminish secretion. With her influx of work and worry there is increased loss of heat, because hitherto she has been lying warm in bed. All this means a largely increased need for food; does she get it? Are we sure that she has had sufficient rest after the mental and physical strain of her confinement? If we could keep the working-class women a few days longer in bed (and in this respect the provision of "home helps" might be useful) might we not be doing something towards the prolongation of breast-feeding? Another point: at or about the tenth day, as has been pointed out to me, there is often a slight loss of blood from the uterus; we know that if a menstrual flow occurs during lactation the milk is often altered in quality and diminished in quantity. Is it not possible, therefore, that if this slight loss occurs at or about the tenth day there may be such an interference with the milk production as to add one more factor to those already mentioned?

Then, too, the baby is undergoing new experiences; he is being introduced to a larger world, his mentality is disturbed; possibly he is now in a cot instead of lying warmly in bed beside his mother. Is he kept as warm as he was? Add to all this the effect of two-hourly feeds, and quite possibly of too frequent night feeding (for it is surprising how often in investigating a case of indigestion we find by close questioning that even babies fed quite properly in the day are fed too often at night), and we arrive at a sum which may account, in many instances, for the failure which undoubtedly does occur at about the end of the first fortnight—in many instances, but not in all, for I have met with more than a few cases in which careful inquiry on the lines I have suggested has failed to account for the failure. Is it a failure common to all grades of society, and, if so, is it a stage in a greater affliction, the inability to suckle at all? Are there any women so constituted? Budin states that there are, and gives an example in his book *The Nursling*. I have never, in my private practice, met with such a case in a woman with normal breasts and nipples, but in my work as medical officer to infant welfare centres I have come across so many women who have solemnly assured me that they have never been able to suckle a child from the first day onwards that I am constrained to believe that there may be a very few cases of this type; in many of these, however, it might be found that the suction of an older and more robust child would be a solution even of this difficulty; and it is significant to note that in the case described by Budin he states that the nipples were "umbilicated," a physical condition rendering it almost impossible to apply efficiently the necessary suction stimulus.

LITERATURE.

Cameron: The Suction Difficulties of Young Infants, *Lancet*, May 13th, 1922, p. 936.
Budin: *The Nursling*, 1907.
Pooler: The Effect of Income on Nursing Capacity and Infant Growth. Report of National Conference on Infant Mortality, 1914, *Midland Medical Journal*, November, 1914.
Idem: Infant Mortality and the Education of the Mother, *Lancet*, July 6th, 1918, p. 3.
Idem: Annual Reports of Birmingham Infant Health Society, 1908-16.
Reports on Infant Mortality, Birmingham Health Department, 1909-10-11.
Pritchard: The Food Requirements of Infants, *British Journal of Children's Diseases*, February, 1914, p. 49.
Pritchard, Carter, and Pitt: Breast-Feeding—the Value of the Test Feed, *Lancet*, September 2nd, 1911.
Forsyth: Observations on Breast Feeds, *Lancet*, June 24th, 1913.
Elgood Turner: Human Milk and Breast-Feeding, *British Medical Journal*, October 15th, 1910.
Report of International Conference on Infant Welfare, Berlin, 1911: Leven and Barrett on X-Ray Examination of the Infant Stomach.
Waller: Clinical Material at an Infant Welfare Centre, *Maternity and Child Welfare*, September, 1919, p. 1.

THE RELATION BETWEEN TRAUMA AND TUBERCULOSIS:

*FROM THE PHYSICIAN'S POINT OF VIEW.**

BY

NORMAN TATTERSALL, M.D.LOND.,
Chief Clinical Tuberculosis Officer, Leeds.

IN discussing the relationship of trauma and tuberculosis from the point of view of the physician I intend to confine my remarks almost exclusively to pulmonary tuberculosis, but there are two other types of tuberculosis associated with injury which may first be briefly mentioned.

1. *Inoculation tuberculosis.*—This is probably the only true form of traumatic tuberculosis, and includes such conditions as butchers' and pathologists' warts, also those cases in which a tuberculous condition follows a cut by a broken sputum flask or other infected agent. Such cases are uncommon, but are true examples of traumatic tuberculosis, as they involve injury to the skin and the introduction of the tubercle bacillus from without. Their pathogenesis is so straightforward that they should not lead to any difficulty from the accident insurance standpoint.

2. *Acute miliary tuberculosis.*—This may occur as an immediate sequel to such injuries as a blow on a tuberculous testis, forcible movement of an old tuberculous joint, or operative measures on tuberculous glands, etc. In such cases it is obvious that the trauma has caused rupture into the blood stream of a tuberculous focus, and in spite of the necessary presence of the tubercle bacillus in such a lesion the trauma is still the essentially responsible factor in the ensuing train of events.

Passing to the relationship between pulmonary tuberculosis and traumatism, the first question which arises is the frequency of such occurrence. I have recently surveyed the histories of 300 consecutive cases of definitely diagnosed pulmonary tuberculosis, and find that whilst injury was suggested as a cause in six cases, there were four in which the evidence clearly linked up the onset of symptoms and the fact of injury. Small though this figure may appear, considerable sums of money may be involved owing to the liability of employers under the Compensation Acts, and it is important, therefore, that we should hold definite opinions as to the part played by trauma in such cases.

Dr. Parkes Weber, writing on this subject in 1910, grouped the cases into:

(*a*) Those cases in which no tuberculosis was known or suspected to exist prior to the injury.
(*b*) Those cases in which tuberculosis was known to be present before the injury.

This grouping may conveniently be followed, and may be compared with the classification of "due to" and

* Read in opening a discussion in the Section of Tuberculosis of the Annual Meeting of the British Medical Association, Cardiff, 1928.

"aggravated by" service which are so familiar in the phrascology of the Ministry of Pensions.

The two following are examples, recently under my notice, of injury to presumably healthy persons.

1. A finely built man, aged 42, with an excellent health record, received a severe blow in the left axilla from the mudguard of a motor car. He returned to work in three days, but a fortnight later had cough and persistent pain in the side. Four weeks after the accident he left work feeling weak and ill. I saw him the following week and found hardly any signs except fine crepitations in the axilla. A radiogram showed a mottled fan-shaped opacity extending from the root to the periphery. The sputum was positive, and after an acute course he died in six months. There was no haemoptysis at the time of the accident or later.

2. An ex-sergeant, aged 36, playing Rugby football regularly and in full training, during a game in December, 1926, was heavily tackled, another player falling with his knee on the back of the patient's right shoulder. After temporary attention he finished the game, but the same evening had slight haemoptysis. He had slight cough and sputum for the rest of the winter, and "never felt fit enough" to play football again. The cough disappeared in the summer, but returned in September, 1927. Finding his "wind was poor" he did not resume football last winter. In January, 1928 (thirteen months after the injury), he had severe haemoptysis, and I saw him for the first time. There were marked signs in the right lung, slight on the left, and a positive sputum. He is still in a sanatorium, doing very well, the disease being of a chronic fibrotic type.

Note the very acute course of the first case and the slow development of the second. In each case symptoms started very soon after an injury and formed a continuous chain of evidence up to the time of diagnosis.

The question to be answered is whether the accident has (1) *localized* a tuberculous lesion at the site of injury, or (2) *mobilized* and activated an old tuberculous focus.

It has frequently been stated that injury produces in the tissues a state of lowered resistance on which is implanted a tuberculous infection, the tubercle bacilli being carried by the blood or lymph stream from some distant and possibly quiescent focus. There is, however, very little evidence that this ever occurs. Animal experiments on this point have only in rare instances produced positive results, nor does clinical evidence support this theory, as is instanced by the fact that accidental or operation wounds in tuberculous subjects heal quite readily.

Recent work by Opie and others shows that tubercle bacilli can be isolated from lung tissue or glands which show no naked-eye evidence of tuberculosis. If apparently healthy tissues so frequently harbour tubercle bacilli this theory of diminished local resistance can hardly be supported, as otherwise local traumatic tuberculosis would be much more frequent than is actually the case. It would appear, therefore, that in cases such as those quoted the injury has directly or indirectly affected an existing focus of disease. Probably in the acute case a caseous root gland ruptured directly into a bronchus, and in the latter a latent apical focus was compressed or torn, disseminating the previously localized infection and leading to slow progression of a previously dormant lesion.

If this be true the difference between injury to the presumed healthy and the known tuberculous subject largely disappears and becomes one of degree only. Further, we know that *post-mortem* statistics yield evidence of latent or healed tuberculosis in 33 to 97 per cent. of cases, and this might well become 100 per cent. if recourse were made to animal inoculation. One German writer laconically observes: "after all, everyone is a little tuberculous."

When injury affects the known tuberculous subject our knowledge is more exact. Rest is still the bed-rock of treatment, and evidence of aggravation by undue exertion is frequent. Tuberculous disease produces physical changes in the chest which favour the transmission of external impulses to the affected area. Injuries—especially those involving compression, and which normally are resisted by the elasticity of the chest wall and contents—will be focused and will exert their greatest influence at that spot where adhesions or loss of elasticity of lung interfere with the normal mechanism of safety.

A point of some importance is that the *post-mortem* findings prove that after severe injuries the lung opposite to the side receiving the impact may show extensive damage to, and even tearing of, healthy lung. Thus an impulse applied to one side may readily aggravate disease in the other lung, especially when adhesions, etc., favour the concentration of the stimulus at the affected spot. It is obvious, therefore, that injury to the chest of a tuberculous subject may readily result in active manifestations of any form of pleural or pulmonary tuberculosis.

Nature of Injury.

Most of the recorded cases of traumatic pulmonary tuberculosis have followed injuries involving compression, such as crushing by falls of coal, etc., or the impact of a large object against a considerable area of chest wall.

The infrequency of pulmonary tuberculosis after war wounds has been recorded by many observers. This may in part be due to the subjects being picked healthy men, but the fact that the passage of a high-velocity bullet through the chest will not produce the diffuse impulse of a crush may be an important factor. The intensity of the injury is probably less significant than its nature, especially as regards the element of pressure.

Injury to distant parts of the body may, through sepsis, etc., so debilitate a patient as to favour the lighting up of a previously latent lesion.

Clinical Aspects.

The course of disease subsequent to injury may be acute, subacute, or chronic.

Haemoptysis and pleurisy are symptoms of great importance, though neither is essential in establishing a claim for compensation. Their importance lies in the fact that they will assist in fixing a date of onset of symptoms, will usually lead to the seeking of medical advice, and haemoptysis, in particular, will not only impress the patient, but, if observed by others, may be valuable corroborative evidence.

In every case most careful history taking is essential, as in the absence of dramatic symptoms the establishment of a claim to compensation may rest entirely on slight but persistent evidence of ill health which bridged the interval between injury and diagnosis. Bridge symptoms of this kind were well shown in the case of the footballer already quoted.

X-ray evidence may be valuable in demonstrating an old and possibly calcified lesion with evidence of recent acute spread. In several acute cases I have observed a fan-shaped area extending outwards from the root, suggesting the rupture of a caseous lung or root-gland focus into a bronchus, with acute broncho-pneumonic spread.

How shall we assess the particular part played by injury in cases of traumatic tuberculosis?

In most cases it cannot be proved that, apart from the injury, the patient would later on have suffered from tuberculosis, but if the evidence convinces us that trauma adversely affected the existing condition, it must be considered the materially effective cause of the present ill health.

The German accident insurance law allows a patient two years' grace in which to assert his claims, and for "aggravation" of lung tuberculosis, six months, which may be increased in special circumstances. This appears to be a reasonable allowance.

The fact that the patient was "fit to work until the accident" may be claimed as proof of his perfect health; this is a fallacy which is daily disproved in the experience of most of us.

The consideration of the following questions in every case is suggested as an aid to arriving at a decision:

1. Was the patient actively or passively tuberculous before the accident, and if the former, what was his expectation of life?

2. Is the fact of injury definitely established, and was it of such a nature as to be likely to damage an active or quiescent lung focus?

3. What period of time elapsed between the injury and the diagnosis, and was this period covered by "bridge" symptoms?

4. Is the present condition of the patient and the course of the disease such as might be expected if the injury had reawakened or aggravated an existing latent or active focus of disease?