It is realized that the mere occurrence of familial disease due to the bovine bacillus will not prove transmission by humans unless infection from a common source is definitely excluded, and, indeed, it is remarkable that heretofore no instance of this kind has been recorded.

In the absence of proof of transmission to contacts, it is of interest to inquire whether there is any peculiarity of the bovine bacillus or of the disease produced by it which would point to a limitation of spread of the disease by infected human subjects. An outstanding feature of tuberculosis of bovine origin is the high incidence of glandular disease in children in the 0-5 years group, and the disproportionately low incidence of visceral disease in the later age periods. This disparity is not related to the virulence of the bovine bacillus, but suggests that it has a lower invasive power than the human type, so that extension beyond the lymphatic system occurs less readily and so the majority of bovine infections in childhood do not lead to late visceral disease. But when spread does occur beyond the lymphatics the organism shows a high virulence and a tendency to produce multiple lesions and in some cases a marked degree of generalization, similar to that resulting from experimental inoculation in rabbits. This combination of low invasive power and high virulence recalls the cultural characteristics of the organism, and may be in some way related to the latter. Bovine bacilli are dysgonic on all artificial media, and are difficult (as compared with human bacilli) to establish in primary culture, but by subculture they can be induced to grow luxuriantly. Their persistence and tenacity of life when once established in culture is well illustrated in the case of a mixed growth of human and bovine bacilli which, when kept in the incubator for some months, yielded only dysgonic cultures, the eugonic element having apparently died out.²

If it is not accepted that the bovine bacillus has a lower invasive capacity, and has greater difficulty in causing progressive disease in man, is there an alternative explanation of the low incidence of visceral tuberculosis in adults? It has been suggested that mutation of type occurs in the human body, so that an infecting bovine organism after many years in human tissues acquires the characteristics associated with human type bacilli, and becomes manifest as such in chronic pulmonary tuberculosis in adults. Against this it may be pointed out that the type characteristics of tubercle bacilli are remarkably stable, less than 3 per cent. showing divergence in cultural character or virulence from standard types, and no range of transitional strains is found. Further, attenuation does not appear to be related to the age of the patient. For example, a bovine strain isolated at Glenlomond from a case of hyperplasia of the large bowel in a patient aged 58 was found to be of standard virulence, and strains isolated from pulmonary cases where the personal history of tuberculosis extended over periods of three, five, and seven years were also of standard virulence. One strain was isolated from sputum on two occasions with an interval of four years between, and no alteration in virulence was found.

Another point worthy of consideration is the number of bacilli observed in the sputum of cases due to the bovine bacillus. In the earlier cases investigated at Glenlomond it was noticed that the bacilli were present in very small numbers, but this does not hold good for the more recent cases, where sputum rich in tubercle bacilli has been obtained. In one case they were present in enormous numbers, and this virus was grown from the sputum only after many attempts, although easily isolated by animal inoculation. So far as contact infection depends upon the number of bacilli thrown off in sputum, there seems to be no reason why it should not occur as easily with bovine as with human type cases.

CONCLUSION

There is no proof that spread of bovine type tuberculosis by humans occurs.

There are opportunities for contact infection under conditions similar to those which cause spread of tuberculosis due to human type bacilli.

It is suggested that the bovine bacillus has a lower invasive capacity than the human, and produces progressive disease less readily.

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ARTIFICIAL PNEUMOTHORAX

AN AFTER-HISTORY OF 113 CASES

BY

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In a recent paper on the effect of fluid complicating artificial pneumothorax the after-history of forty cases was considered in detail. These forty were exactly half the number of sputum-positive cases in which I was able to induce a pneumothorax between 1924 and 1930, and in which for various reasons refills have ceased. All cases have been followed up to November, 1933, so it is now possible to compare results (1) in forty cases that did not develop fluid and forty that did, and (2) in the total eighty cases with "successful" inductions, and thirty-three "control cases" where induction was not successful. At the time when induction was decided on, each case was allocated to a "choice" I, II, or III in accordance with the type and extent of disease, but these subdivisions have not been included in the present summary.

TABLE I.—Cases Successfully Induced: No Fluid. Still Alive

	T.B. +	T.B. - and No Sputum	Totals
Completed treatment ...	4	21	25
Re-expanded	1	2	3
Refills stopped for fresh disease in other lung	2	3	5
Totals	7	26	33

Thirty-three of the forty cases that did not develop fluid during the treatment are alive, and twenty-six of these are sputum-free or have negative sputum. Four of these had laryngeal tubercle: all four are soundly healed. Under the heading "completed treatment" are given those in whom refills were carried on for at least three years and until such time thereafter as it was considered safe to re-expand the lung. Four of twenty-five in this category are now sputum-positive; two became positive again soon after re-expansion, and in both phrenic evulsion was performed, while one of them also had sanocrysin, but both have developed signs of disease in the better lung. The other two became positive at intervals of a year and two years after the re-expansion.

"Re-expanded" cases are those where for no apparent reason the lung re-expanded during the treatment by refills. Of three such patients two again became sputum-positive; one refused any surgical intervention, and has now extensive bilateral disease; the other had severe haemoptysis, and Mr. Tudor Edwards performed thoracoplasty with such success that the patient has been symptomless and fit for work for two years.

In five cases refills were stopped because fresh disease appeared in the better lung; two remain sputum-positive;

of the three who have again become negative two had phrenic evulsion and one sanocrysin.

Seven of the forty patients are dead. In six the immediate cause was extension of disease; one of these was treated with bilateral pneumothorax for a year; though the larynx healed the patient went steadily downhill. Only two of these six cases had become sputum-negative at any time during the treatment. The seventh died of lymphadenoma.

Thirty-three cases of "controls" have been followed up over the same period. These are cases which were chosen for pneumothorax on the same criteria as the eighty where induction was successful but where induction was not possible as no free pleural space or only a small pocket was found, or where a pneumothorax could not be maintained beyond a few refills owing to the presence of adhesions too gross and too numerous to justify submitting them at that time for cauterization.

TABLE II

	Alive		Dead	Totals
	T.B. +	T.B. - or No Sputum		
No space or pocket only...	6	4	11	21
Adhesions	—	1	11	12
Totals	6	5	22	33

Table II shows that the prognosis in these "control cases" is grave, and is much more so in those cases with adhesions as against those with no free pleural space, as only one of twelve is still alive as against ten of twenty-one. All were treated or advised on treatment by extensive rest, and forms of ancillary treatment were given full consideration. Only three of the twenty-two dead would consider even the minor operation of phrenic evulsion; of the six alive sputum-positive cases one has had phrenic evulsion and one thoracoplasty, and both are in fair condition.

For purposes of comparison the eighty cases where it was possible to induce pneumothorax have been grouped under the heading "successful," and they have been subdivided into those which did and those which did not develop signs of fluid during the treatment, while the thirty-three "control cases" have been labelled "unsuccessful." Table III summarizes the results; it gives the numbers and percentages alive and dead under each subdivision, and among those alive the numbers and percentages sputum-positive, sputum-negative, and fit for work.

TABLE III

		Alive						Dead		Totals
		T. B. +		T. B. - or No Sputum		Fit for Work				
		No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	
Successful	No fluid ...	7	17.5	25	65.0	27	67.5	7	17.5	40
	Fluid ...	6	15.0	25	62.5	24	60.0	9	22.5	40
Unsuccessful		6	18.2	5	15.2	6	18.2	22	66.6	33

From this table the following points emerge:

With a three-year to nine-year after-history,

1. Of eighty "successful" cases, sixty-four (80 per cent.) are alive, and of these fifty-one (63.75 of the total) are now tubercle-negative or have no sputum.

2. Of thirty-three "unsuccessful" cases only eleven (33.3 per cent.) are alive, and of these but five (15.2 per

cent. of the total) are now tubercle-negative or have no sputum.

3. Of eighty "successful" cases, fifty-one (63.75 per cent.) are now fit for work, as against six (18.2 per cent.) of thirty-three "unsuccessful" cases.

4. Of eighty "successful" cases sixteen (20 per cent.) are dead; of thirty-three "unsuccessful" cases twenty-two (66.6 per cent.) are dead.

5. The end-results in "successful" cases, with fluid and without fluid, are practically parallel.

OCULAR TORTICOLLIS: INFERIOR OBLIQUE TENOTOMY AND ITS INDICATIONS

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For many years I have been vaguely aware of a syndrome associated with overaction of the inferior oblique muscle of the eye, but it is only during the last two years that I have given the subject the attention it deserves. Duane of New York published an article in the *Archives of Ophthalmology* in 1916, but since nothing, so far as I am aware, has appeared in British literature on the subject, I feel that some further reference based on personal experience may not be out of place.

It may not be amiss to remind the reader that, when looking straight ahead, elevation of the eyes is effected by the action of the superior rectus muscles. When, however, the eyes are directed sideways—for example, to the left—elevation is accomplished by the co-ordinated action of the left superior rectus and the right inferior oblique. This is another way of saying that, for purposes of elevation, when the eyes are directed to the left, the right inferior oblique and the left superior rectus are complementary muscles. The reason for this will be obvious on looking at the diagrams (Figs. 1 and 2). When either eye is directed outwards its long axis lies virtually parallel with the line of action of the rectus muscles. Elevation (or depression) as it affects this (outward-turned) eye will find the rectus muscle in a position of mechanical advantage. When, however, the right eye, for example, is turned inwards to the left, its rectus muscle forms, in relation to the long axis of the eyeball, a very obtuse angle. The superior rectus is now at a mechanical disadvantage as an elevator. Indeed, such action can only be minimal. The task of elevation of the right eye when turned to the left is now relegated to the right inferior oblique, which, it will be noted, now lies with its line of action parallel with the long axis of the eye.

ALTERNATIVES: OCULAR TORTICOLLIS OR SQUINT

It would appear that weak action (paresis) of a superior rectus is not very uncommon. Whether it is due to a congenital defect or to a birth injury I do not know; I suspect the latter. The mechanism of the co-ordinated movement referred to above is a delicate one, and when it is deranged the following is what I conceive happens. For the movement of the eyes upward and, say, to the left, a certain amount of nerve energy is available. That moiety which would normally act on the left (weak) superior rectus cannot now act with the usual effect, and so overflows to the other component, the complementary right inferior oblique, causing overaction. This hypertrophies, the initial diplopia becomes accentuated, and the overgrown muscle habitually overacts, resulting in the pronounced upshoot of the eye characteristic of the condition when looking laterally upwards.