Further, the suppurative cases tend to run a subacute or almost chronic course. The patient is also exposed to the risk of the ordinary complications of suppurative otitis media. Lermoyez considers that mastoid suppuration is an almost invariable accompaniment of these otorrhœas. The degree of severity of the typhoid fever seems to have no bearing on the occurrence of otitis media, nor on the gravity of the otitis when it does occur.

The usual route of infection is from the naso-pharynx *viâ* the Eustachian tube. Bucco-pharyngeal ulceration, dryness of the mouth interfering with deglutition and diminished resistance of the organism are all factors in the causation. Pre-existing pharyngeal and nasal lesions are predisposing causes.

Treatment is, in the first place, prophylactic—gargles, antiseptic washes, etc. Any pharyngeal lesions which appear should be energetically treated. Once middle-ear suppuration has occurred it should be dealt with on ordinary principles. Any pre-existing nasal or pharyngeal trouble should be attended to as soon as the patient's general condition permits.

(3) Otitis interna is a rare complication. It occurred in only one of the 359 cases. Attacks of slight severity no doubt occur often, but are not recognised owing to the debilitated state of the patient. This complication arises when the fever is at its height, and may be due to hyperæmia, hæmorrhage or serous effusion in the labyrinth.

The only suggestions for prophylactic treatment are the avoiding of quinine and salicylic acid. For the lesion itself, once it has occurred, Hill recommends iodide and mercury with subcutaneous injections of pilocarpine. *John M. Darling.*

MISCELLANEOUS.

Feldt, A.—The Treatment of Tuberculosis with Gold. "Deutsch. med. Woch.," No. 12, 1913.

On injection into animals cantharidin causes a local reaction in the shape of a serous infiltration at any existing inflammatory focus, whether tuberculous or otherwise. It was suggested to the author by Prof. Spiess, who had observed this reaction in the human larynx, that cantharidin, although itself without bactericidal power, might be employed to convey substances possessing such power through the blood-stream to the tuberculous focus.

It was necessary in the first place to reduce the marked toxicity of cantharidin, and this was accomplished without diminishing its affinity for tuberculous foci, by forming a new æthylendiamine compound. The latter was then combined with various salts of gold, which are said to be the most powerful destroyers of tubercle bacilli at present known, their virtue being due, as the author was able to prove, to the gold itself and not to the substances with which it unites to form salts.

Animal experiments carried out with the compounds thus obtained gave the following results. Guinea-pigs and rabbits about a month after injection with either human or bovine bacilli showed on subcutaneous or intravenous injection of the gold-cantharidin compound a very marked local reaction of all the affected organs. The author attributes this reaction to destruction of the bacilli in the periphery of the tuberculous foci with liberation of their contained toxins—in fact, a tuberculin reaction resulting from bacterial destruction.

This focal reaction following injection of gold preparations may therefore be termed the "secondary tuberculin reaction."

Referring to the curative results of his experiments on guinea-pigs and rabbits, the author states that intravenous injection in tuberculous rabbits not only prolonged life for many months, but even brought about a permanent increase of weight. The animals received seven injections during a period of two months. They were killed five months later and the lungs then showed healed tuberculous foci.

The result in guinea-pigs treated by *subcutaneous* injection was negative, the gold preparations being reduced and the gold fixed locally, so that it failed to reach the bacilli in sufficient concentration. The injections also caused much local necrosis. A further hindrance to prolonged treatment with gold preparations consists in the observed fact that the bacilli after a time become "gold-fast," and therefore much less easily destroyed by the compounds in question.

If the latter can be combined with some agent which will overcome this acquired insusceptibility, a satisfactory remedy for tuberculosis may be the result.

Clinical observations will be given later by Spiess at the forthcoming International Congress of Medicine. *Thomas Guthrie.*

REVIEWS.

The Causes leading to Educational Deafness in Children, with Special Reference to Prevention. By Macleod Yearsley, F.R.C.S.Eng. London: P. S. King & Son, Orchard House, Westminster, 1912.

This pamphlet, the reprint of a series of articles published in the *Lancet*, consists of a careful statistical analysis of the histories of no fewer than 2197 deaf children. In the past, the author reminds us, statistical inquiry of this description has been vitiated by the fact that nearly all the data have been made up of the observations of lay, and very often uneducated people, and Yearsley's first care has been to strengthen his foundations by checking with his own observations "the replies given by parents." In spite of this, however, as the author himself says, much of the information supplied is of questionable value. If, for example, we turn to the group entitled "congenital deafness," we find that the numbers given largely depend—as, indeed, they must depend—upon the impressions of people who have never been trained to observe accurately, and that, moreover, these impressions concern the date when deafness is first noticed in an infant—and this even expert observers often find difficult to determine. Indeed, it is not too much to say that the very fact of "congenital deafness" might even be altogether doubted were it not that the existence of other deaf relatives in a family proves its reality as a hereditary defect appearing probably at an early stage in the development of the individual. We hasten to add that this difficulty is quite clearly appreciated by the author (p. 7).

We should like at this point to draw attention to the expression "hereditary (or congenital) deafness," as a term devoid of precision. Science no longer speaks of "hereditary *blindness*," or "congenital *lameness*." The fact is, of course, that we are here faced with a lacuna in our pathology. What and where is the inherited lesion which induces deafness? In short, despite several suggestions and investigations, this branch of otology is still shrouded in darkness.

With regard to the group of "congenital deafness" in which the family history shows no other deaf-born relatives, but contains instances