constituents of the faeces, appears to be gathered into the white layer by a process of sedimentation. During the precipitation and sedimentation of the organic matter the bacteria are carried down, perhaps in some degree agglutinated. This concentration of the organisms makes up for the disadvantage caused by the initial dilution of the material.

For microscopic examination a drop is then taken from the white curdy layer by means of a pipette and placed at the end of a slide, along with a drop of albuminous water from a mixture containing 1 part of egg albumin to 10 of distilled water and 1 per cent. of formalin, and 90 per cent. of distilled water is added. A film is made in the usual way by drawing out the fluid with a second slide along the surface of the first. The albumin, which is recommended by Seeman,⁵ helps to produce a better film and prevents the film from being washed off.

As to the strength of antiformin to be used, we have found that 15 to 20 per cent. is most suitable. The use of a stronger solution, say 50 per cent., is apt to lead to separation of the film. The time during which the action of the antiformin may be continued does not require very strict limitation; although Seeman says that the bacilli cannot stand its action for twelve hours, we have not been able to determine any deterioration of staining properties of the bacilli after twenty-four hours' action when use is made of the 15 to 20 per cent. dilution. The action of the antiformin seems to be chiefly exerted during the first hour, so that there seems no reason to continue the process for more than that time. Indeed, if the white layer has formed well within half an hour, the film may be prepared then. Even when the curdy layer does not become quite white, but remains of brownish tint and small in amount, our experience is that little is to be gained by waiting longer than an hour. The excess of brownish coloration is commonly dependent on an excess of faecal matter, or, perhaps, on the presence of iron taken as medicine. The prepared slides are fixed by heat and stained in the usual way by carbol-fuchsin and methylene blue, being decolorized with both acid and alcohol. As the tubercle bacilli are commonly sparse, it is advisable to prepare two or three films from each specimen.

Microscopically the film is found to consist entirely of a blue-stained residue. Here and there tubercle bacilli are conspicuous by their reddish colour. In most instances they require much searching, as their total number is small. Only in two of the considerable series of specimens examined by us could they be said to be abundantly present.

Some doubt might be suggested as to the identity of the bacilli. Willson and Rosenberger were able to distinguish the bacilli in the stools examined by them by means of prolonged exposure to Pappenheim's stain. Other acid-fast bacteria, with the exception of the bacillus of Johne's disease, lost their carbol-fuchsin stain in twenty minutes. Tubercle bacilli retained it for one hundred and sixteen days.

Our slides were decolorized with alcohol as well as acid. Bacilli were not present in the faeces from the normal cases examined. The bacilli in both sputum and faeces possessed the same characters. One case with short, thick bacilli in the sputum had short, thick bacilli in the faeces.

We attempted to make cultures from the faeces, but a large number of resistant spores occur in faeces, and we were unable to destroy them without also destroying the tubercle. While it is possible to obtain pure cultures of the tubercle bacillus from sputum, faeces present greater difficulties, and so far our attempts have proved unsuccessful.

The results obtained by us may be summarized as follows. Altogether 109 different specimens were examined. Of these, 99 were from patients suffering from pulmonary tuberculosis, in different stages of the disease, in the Royal Victoria Hospital for Consumption. None of the patients had intestinal tuberculosis. The others were from patients in the Royal Infirmary, one from a case of tuberculous peritonitis, and the others from patients without a suspicion of tuberculous infection.

Of the 109 persons, only 34 had sputum in which tubercle bacilli had been discovered; 42 had sputum in which no bacilli were found, and 24 had no sputum whatever.

Of the 100 tuberculous cases examined 79 were found to yield tubercle bacilli, and only 21 were negative. Of the 9 specimens from presumably normal subjects none yielded tubercle bacilli.

Of the tuberculous patients, all those with bacilli in their expectoration, with one exception, presented tubercle bacilli in the faeces. Of the 42 cases in which bacillary examination of the sputum was negative, 29 gave tubercle bacilli in the faeces. Of the 24 cases without sputum 17 showed bacilli in the stools.

These results are striking, and are suggestive in regard to clinical diagnosis, and to the larger aspect of preventive medicine. Thus it may be stated with absolute certainty that the occurrence of tubercle bacilli in the faeces is independent of the presence of an intestinal lesion. Indeed, their presence has little reference to abdominal tuberculosis. They were actually absent from the only case of tuberculous peritonitis which was examined.

The remarkable frequency of their occurrence in relation to pulmonary tuberculosis, not only in cases where tubercle bacilli were determinable in the sputum, but also in cases where either bacilli were absent or there was no sputum whatever, would seem to afford an important addition to our means of diagnosis in doubtful cases of pulmonary tuberculosis.

From the public health point of view, the almost unfailing presence of tubercle bacilli in the faeces—the presumption is that in some of the negative cases in the present series more minute examination would have discovered them—raises questions as to the need for disinfection of stools, more especially in the case of bed-ridden individuals, such as has hitherto not been thought necessary. Further, if, in the Royal Victoria Hospital where swallowing of sputum is carefully guarded against, tubercle bacilli are found to occur so constantly in the stools of the patients, whether expectorating bacilli or not, it may be assumed that in the great proportion of untreated cases, where swallowing is often the rule, the number of tubercle bacilli discharged in the faeces will be vastly greater.

It may be added that the technique necessary for the clinical detection of the bacilli is not difficult or lengthy, and that the process of faecal examination, which is apt to be repellent to some observers, has most of its unpleasantness removed by the method which has been described.

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THE EXPERIMENTAL EVIDENCE FOR THE THEORY OF THE NEUROGENIC CO-ORDINATION OF THE HEART IMPULSE.*

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IN an abstract of the work of Cohn and Trendelenburg in the *Centralblatt f. Physiol.*, 1910, No. 5, p. 183, doubt is cast upon the observations, made many times in the Hallerianum Berne, which speak for the neurogenic nature of the conduction of the heart impulse. The authors have obviously not had time to study the work on this subject, and yet pronounce the opinion that the fundamental experiments of Pauk were carried out "with entirely insufficient methods." In August, 1907, Pauk communicated and demonstrated his experiments to the Seventh International Physiological Congress in Heidelberg.¹ Not one of the experts present doubted the result.

The myogenic theory does not explain how it is that the ventricles can fibrillate while the auricles pulsate, and vice versa; or why a thrust in the right spot into the intraventricular septum brings the heart into fibrillation; or why, what is easier, paraffin injected into the peripheral twigs of the coronary arteries produces a like

* See Appendix D, pp. 224-227, in Sir Lauder Brunton's *Treatise on the Circulation* (John Murray), 1908.

effect.³ Under these circumstances, according to this theory, we must suppose that one part of the auriculo-ventricular bundle is fibrillating and the other beating.

On March 18th, 1884, I found in the dog's heart, close under the descending branch of the left coronary artery, a nervous bridge, the ligaturing of which immediately destroyed the co-ordination of the beat between auricles and ventricles. The auricles maintained their frequent beat (160 per minute), while the ventricles beat only 40 to the minute. Professor His examined the ligatured bridge and pronounced it to be a nerve bundle and a group of ganglion cells.⁸

In the rabbit Lomakina saw the auricles and ventricles beat quite independently after separate ligations had been made of the aorta and pulmonary artery.⁴ In a dog a ligature applied 4 mm. above the sulcus produced fibrillation of the left auricle, a quickened beat of the right auricle, and a less frequent beat of the ventricles. After loosening the ligature, co-ordination again returned. When the aorta and pulmonary artery were again ligatured, the auricles beat more quickly, the ventricles more seldom. Ligation of the whole heart above the auricles caused these to cease beating, while the ventricles continued. In another dog the right and left auricular appendices were clamped. Both auricles stopped, the ventricles beat. A strong tetanic current upon the left auricular appendix induced 132 beats a minute in the auricles, 104 a minute in the ventricles; strong vagus stimulation, 184 beats in auricles, 80 in ventricles. In a rabbit ligature of the root of the pulmonary artery caused 148 auricular beats, 92 ventricular beats; later 140 auricular to 72 ventricular. Heating the nerve plexus at the base of the great arteries acted even more intensely than ligation; one or both auricles ceased to beat, while the ventricles either continued to beat or fibrillated. After injury or destruction of the plexus situated at the base of the left auricle, we observed entirely different movements of both ventricles to those produced by the ligation experiments. Distant effects were also obtained, for after coagulation of the nerves at the root of the pulmonary artery the base of the left auricular appendix beat before its apex.

Professor Busch (Buffalo) found in the Hallerianum⁵ that the apex of the rabbit's heart no longer pulsated if it were cut away from the ventricular base until connected only by a moderately broad bridge of tissue. The extirpated dog's heart beat if perfused through the left descending coronary artery with blood half diluted with saline. If tetanized it fibrillated only during the period of stimulation. If normal dog's blood were used for the perfusion and the beating heart was then tetanized, the fibrillations became lasting, but could be removed by the perfusion of diluted calf's blood. A strip of ventricle connected to the base by muscle and the coronary vessels beat more seldom than the rest of the heart. If fibrillations were induced in the heart the strip participated, but began to beat when the vessels were ligatured. In one case the bloodless strip fibrillated also.

Dr. Imchanitzky-Ries proved microscopically in 1905 that quickly fixed pieces of the pulsating ventricles showed an equidistant transverse striation everywhere, whereas pieces of the fibrillating heart showed fibres in different stages of contraction.

The phase of contraction, therefore, does not pass beyond the bounds of the fibre. Inside one fibre also the transverse striation can be different.

This discovery proves that the muscle fibres possess no direct functional connexion. As in the beating heart the fibres all show the same phase, the connexion must be independent of the muscle—that is to say, through the nerves.⁶

At Palermo I found with Spaletta in the heart of the turtle a nervous bridge running on the outer side from the auricles to the ventricle. Section of this bridge destroyed co-ordination.⁷ In hibernating animals (rectal temperature of 5° to 8° C.), with the heart beating 12 to the minute, the strongest induction shocks did not produce increase in the rhythm, whereas moderate shocks in the awakened warm-blooded animal would produce fatal fibrillary contractions;⁸ consequently the muscle is not directly excitable.

Paukul in a series of experiments carefully ligatured the auriculo-ventricular bundle (the bundle of His). Microscopic examination confirmed the crushing by the ligature. Nevertheless, the auricles and ventricles con-

tinued to beat with the normal rhythm. Coloured plates of the sections of the bundle have been published. Ligature of the whole heart between auricles and ventricles induced allorhythmia. In one case, too, the introduction of the needle near the bundle without ligaturing produced the same effect. Of this the curves and microscopic evidence have been published.

Cohn and Trendelenburg have cut the auriculo-ventricular bundle in very exact fashion in cats, rabbits, dogs, apes, and goats; they find always that co-ordination was abolished. But these experiments were made not on the living but on the perfused or surviving heart. Langendorf found that perfused cats' hearts will still beat after they are cooled to 6° C. The living dog's heart ceases to beat at 25° C.

It is not necessary to perfuse the coronary arteries of the fibrillating or resting heart with blood to make it pulsate again. Cutting out, as I showed in 1884, suffices; then it pulsates as a whole or in sections either spontaneously or on stimulation.

Our results apply to the living intact heart.

Imchanitzky-Ries found in the lizard (*Lacerta ocellata*) that the chief nervous connexion between auricles and ventricle is situated on the dorsal side exteriorly. It contains also many ganglion cells. Ligature of this plexus destroys the co-ordination of the auricular and ventricular beat.⁹

From these observations it follows that the co-ordination of the different parts of the heart is brought about through a nervous channel. Even if the surmise of Cohn and Trendelenburg were correct, that Paukul had not cut all the fibres of the auriculo-ventricular bundle when he saw the auricles and ventricles beating in co-ordinate fashion, both his observations and ours of the inco-ordinated rhythm induced without any interference with the bundle prove that the auriculo-ventricular bundle is not necessary for co-ordination or does not suffice.

In this paper I have given only the observations made by myself or my collaborators.

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NOTES ON TWENTY-EIGHT CASES OF MONGOLIAN IMBECILES:

WITH SPECIAL REFERENCE TO THEIR OCULAR CONDITION.

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THE following characteristic features were observed in the study of 28 cases of Mongolian imbecility at present resident at Earlswood Asylum. Before discussing them it should be noted that, as judged from 50 consecutive cases, the lives of Mongolians are comparatively short. At Earlswood, the average age at death has been 14 years 7 months for boys, and 14 years 6 months for girls, as judged from 21 consecutive cases. Death was due to the tubercle bacillus in nearly 100 per cent. of the cases. This is clear from the following table:

Cause of Death.	Males.	Females.
Pulmonary tubercle	29	12
General tubercle... ..	8	5
Tabes mesenterica	5	2
Acute miliary tubercle	1	1
Pneumonia	4	—
Bronchopneumonia	1	1
Morbus cordis	1	—
Erysipelas	1	—
	50	21