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ADENOVIRUS INFECTIONS OF THE EYE

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The study of viral agents in human diseases is a very dynamic field where very great strides have been made in the last 15 years mainly because of improved tissue culture techniques. The purpose of this paper is only to give a brief review of adenoviruses in general and of infections of the eye with this virus in particular.

Virus infections of the eye may be primary as in trachoma, inclusion conjunctivitis and epidemic kerato-conjunctivitis. On the other hand, the eye may be involved secondarily in a general viral disease as in small pox, measles and pharyngo-conjunctival fever.

The group of viruses now known as adenoviruses was discovered by two independent groups of workers in U. S. A. Huebner, Rowe and co-workers [1] while growing adenoids and tonsils in tissue culture, found that in some the cells, after growing well for a few days, started degenerating. Suspecting a viral agent, they passed the fluid from these cultures into cultures of other indicator cells which showed a characteristic "cytopathic" effect. As this effect was easily repeated in further passages, they called this virus "adenoid degenerating agent".

Another group, led by Hilleman of Walter Reed Institute in Washington, was investigating the respiratory diseases prevalent among U. S. army recruits[2]. They encountered a similar agent which they called ARD 61. When both groups found that they were dealing with the same agent, these were called APC (Adenoidal-Pharyngeal-Conjunctival) viruses. In 1956, it was decided to give this group of viral agents the name adenoviruses[3]. So far 29 serotypes, including 5 isolated from monkeys, have been identified. Infections of the eyes, throat, lymph nodes and lungs with these viruses have been reported from all parts of the world. Though these were isolated and identified only in 1953, a considerable volume of literature has grown round this group. Workers looking for a harmless agent to study the fundamental nature of viruses have found in this group a very handy tool.

The most important feature of these viruses is that they are not transmissible to any of the laboratory hosts commonly used in virus work like the embryonated egg, mouse, rabbit, etc. These agents thus became apparent only when cell cultures became feasible in ordinary laboratories.

The adenoviruses are not inactivated by ether or antibiotics. This feature is important as all media used for cell culture contain penicillin and streptomycin. In size they are about that of the influenza virus (100 millimicrons); they are more stable than the influenza group.

This group of viruses has an affinity for lymphoid tissue. Adenitis of the draining glands is often found in pharyngo-conjunctival fever and eye infections caused by these viruses. Table 1 gives a summary of the various clinical conditions caused by infection with these viruses.

TABLE I.

Association of Adenovirus Types with Human Illness.

	Adenovirus Types	
	Common	Less common
Acute undifferentiated respiratory disease ...	4, 7	3, 14
Acute febrile pharyngitis ...	1, 2, 3, 5	
Pharyngo-conjuctival fever ...	3, 7a	1, 2, 5, 6, 14
Follicular conjunctivitis ...	3, 7a	2, 6, 9, 10
Epidemic kerato-conjunctivitis ...	8	3, 7a
Viral pneumonia		
Infants and children ...	7a	1, 3
Adults ...	4, 7	1, 3
Viruses isolated in Saudi Arabia from cases of trachoma ...	15, 16, 19, 20	17, 21 to 24

When these viruses are grown in a sheet of susceptible cells, the virus particles attach themselves to the cells for a few hours (the eclipse phase); then they penetrate into the cell and multiply in the nucleus and subsequently spread tnemselves in the cytoplasm. At this stage, if a stained cell preparation is examined, intranuclear and cytoplasmic inclusion bodies can be detected. Different types of adenoviruses show varying rates of multiplication. Under the low power of the microscope, infected living cells can be made out by their rounding and shrinking. With good multiplication of virus, vacant areas are found due to the falling away of the cells. This is called cytopathic effect. Infectious virus is found in the cells and also in the supernatant nutrient fluid. By repeated freezing and thawing, the host cells can be made to rupture, liberating more virus into the medium.

In many cases the tonsils and adenoids harbour these viruses. Some workers claim to have isolated adenoviruses from 57% of tonsils and

adenoids surgically removed, by growing these tissues in bottles. From 40 specimens of adenoids processed in this laboratory, we have obtained only 5 strains.

The serological changes, caused by infection with any member of this group are of great interest. Like most other viruses, in the host they produce both neutralizing and complement fixing antibodies. The neutralizing antibodies are type specific. The diluted serum is mixed with a measured quantity of the known type of active virus, and the mixture is introduced on a sheet of susceptible cells in a tube. If there is neutralizing antibody to that type in that serum, there will be no cytopathic effect. In the absence of the antibody, the virus shows cell destruction. Facilities for cell culture are essential for doing this test. The C. F. test, on the other hand, can be done in any serology laboratory if the antigen is available. After performing C. F. tests for adenoviruses on 2308 sera received for other serological tests in this Institute, it is found that 27% show the presence of C. F. antibodies in a titre of 16 and above pointing to a fairly wide spread infection with this group of agents.

Pasteur Institute, Coonoor is one of the few laboratories in this country working on this group of viruses. Table 2 shows the number of strains isolated here with their sources and types. We have used human amnion cells[4] mostly for our isolation and studies. HeLa cells also have been used.

TABLE II.

Adenovirus Strains Isolated in Coonoor.

Year	Types					Undetermined	Total
	1	2	3	5	7		
			A. THROAT SWABS				
1958	...	...	...	...	1	...	1
1959	...	3	...	...	1	1	5
1960	1	2	2	...	...	3	8
1961	1	...	...	...	...	5	6
	B. TONSILS AND ADENOIDS SURGICALLY REMOVED						
	3	1	...	1	...	...	5
			C. CONJUCTIVAL SCRAPING				
	...	...	1	...	...	1	2
							27

In Table 1, the different clinical conditions of the eye, caused by adenoviruses are given. Epidemic kerato-conjunctivitis, caused by type 8 adenovirus is the most important of these, as extensive epidemics have been reported, in recent years, from U.K., Japan, Hawaii, India, Indonesia

and Italy. The enlargement of the preauricular lymph nodes in adenovirus infections of the eye seem to be helpful in arriving at a diagnosis.

In follicular conjunctivitis, which resembles trachoma in several respects, types 3, 7, 15, 16, 19 and 20 have been isolated. Workers in Saudi Arabia [5] have isolated for the first time 6 new serotypes from cases of chronic follicular conjunctivitis. During the three years 1954 to 1956, they inoculated 948 conjunctival scrapings from suspected cases of trachoma into tubes with growing human conjunctival cells. They isolated 65 strains of adenoviruses (7%). Though they belonged to 13 different serotypes, the most common were types 3, 8, 15, 16 and 17. The most interesting finding was that 92% of isolations were made in summer and that 90% of these were from children below 3 years. The general finding appears to be that there is a greater immunity against these viruses than against trachoma virus.

Several experiments of inoculating these viruses into the eyes of healthy volunteers have been reported. Mitsui's work [6] in Japan is of great interest. He selected 5 volunteers with no antibodies in their serum and with no history of conjunctivitis. Clear tissue culture fluid containing type 8 adenovirus was instilled into the right eye of each volunteer; and similar fluid without virus was instilled into the left eye of each. After this they were isolated for 10 days. On the 5th and 7th days, 3 of the volunteers developed acute follicular conjunctivitis with preauricular lymphadenitis. A week later, sub-epithelial keratitis developed in them. In one the disease was very mild. The fifth did not take the infection.

Saudi Arabian workers [7] transported 3 types of adenoviruses after freeze-drying them to an orphanage in Hong Kong and instilled into the eyes of 3 child volunteers. All three developed follicular conjunctivitis on the 4th day, which persisted till the 12th day and then slowly regressed. At the end of a month they developed both neutralizing and C. F. antibodies in their serum. The effect on the cornea could not be followed in these volunteers, as they had abnormalities of the cornea to begin with. Early workers on this group of viruses in U. S. A. (Bell and others) [8] swabbed the palpebral conjunctiva of volunteers with fluid containing adenovirus types 1, 3, 4 and 5. Ninetythree percent of the volunteers first revealed conjunctivitis in 2 to 7 days and then pharyngitis. Serum taken three weeks after the experiment also showed neutralizing antibodies.

The U. S. Naval Research Unit in Taiwan [9] (Formosa), while trying out trachoma virus in volunteers, tried the effect of type 4 adenovirus on one volunteer. The ophthalmologist examining the cases did not know who were given trachoma and who adenovirus. For the first 2 weeks both trachoma volunteers and the adenovirus volunteer showed the same type of follicular conjunctivitis. During the next fortnight, the conjunctiva of the adenovirus patient showed much milder signs and became normal at the end of one month.

Two laboratory infections also have been reported with this virus, one from Saudi Arabia and the other from California. In both cases, the virus was reisolated from the patients.

No specific measures are available now for the treatment of these conditions.

Ophthalmologists and physicians examining the eyes of patients have to be specially careful not to transmit these agents from the infected eyes to the healthy ones by using contaminated fingers and instruments.

Summary: A brief review of the characteristics of adenoviruses and their ocular manifestations is given. The incidence of these infections in this part of the country, based on investigations carried out at the Pasteur Institute, is presented.

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IMPORTANT ANNOUNCEMENT

It is proposed to bring out the November, 1961 issue of our Journal as a special number on 'Neurology'. This number will be very useful to the general practitioners as a hand-book of reference on Neurology. Readers are specially requested to watch for the date of release of this special number — 21st November, 1961 and to preserve this copy for their reference.

MANAGING EDITOR.

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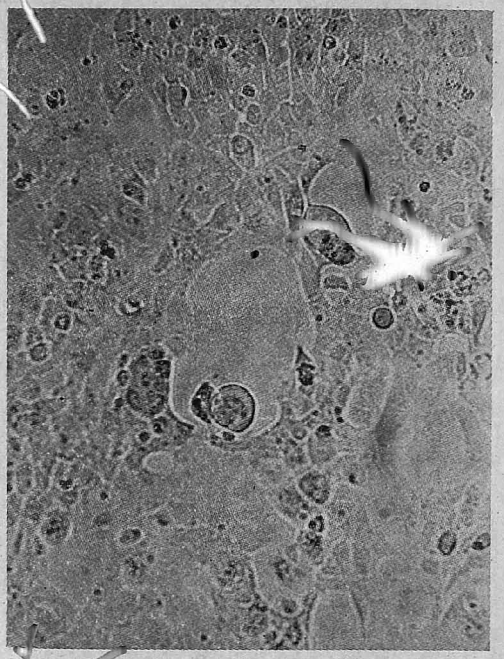

FIG. 1

Microphotograph showing early cytopathic effect caused by type 4 adenovirus in HeLa cells. (Low power)

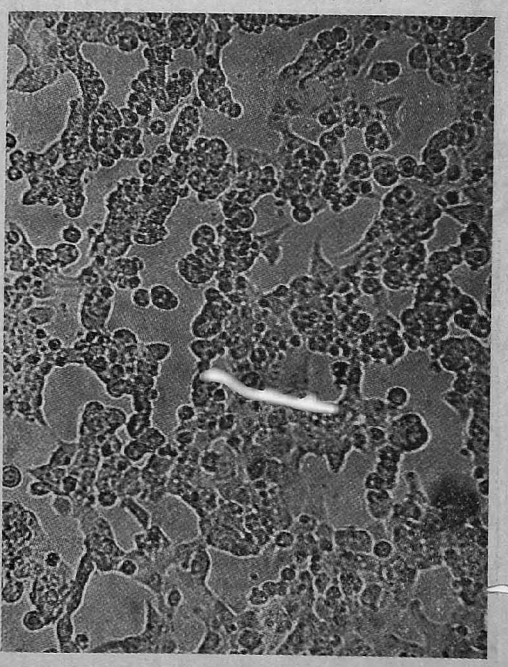

FIG. 2

Microphotograph showing late cytopathic effect caused by type 4 adenovirus in HeLa cells. (Low power)

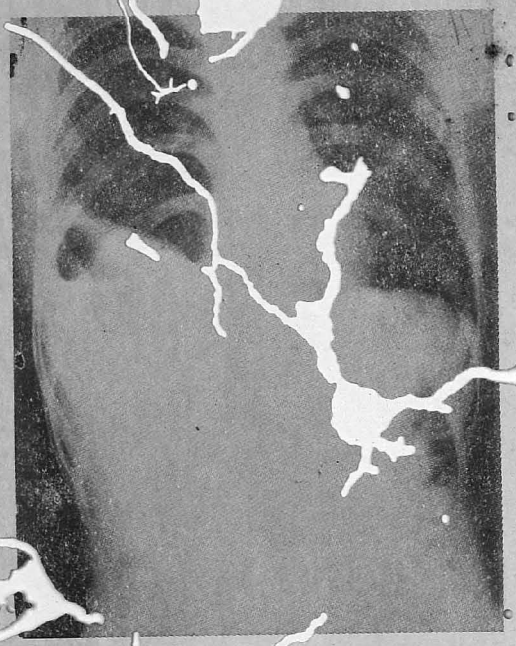

FIG. 1

Amoebic Liver Abscess: Aspirated & air introduced to visualise cavity: P. A. View.

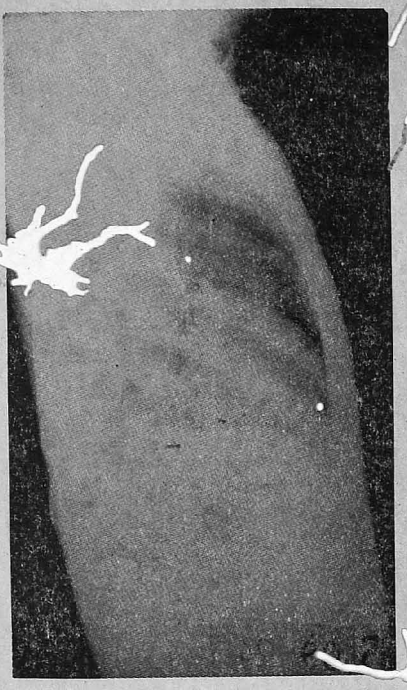

FIG. 2

Lateral view indicating posterior location of the abscess.

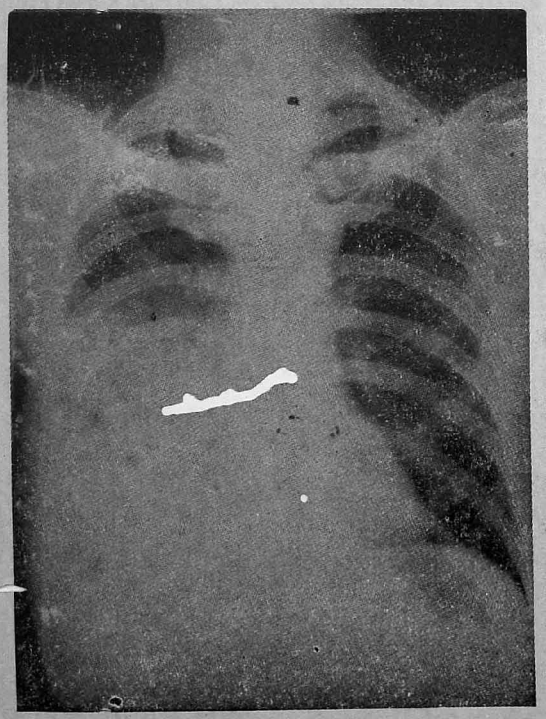

FIG. 3

Empyema Secondary to Liver Abscess: Amoebae in pleural biopsy and pleural fluid.

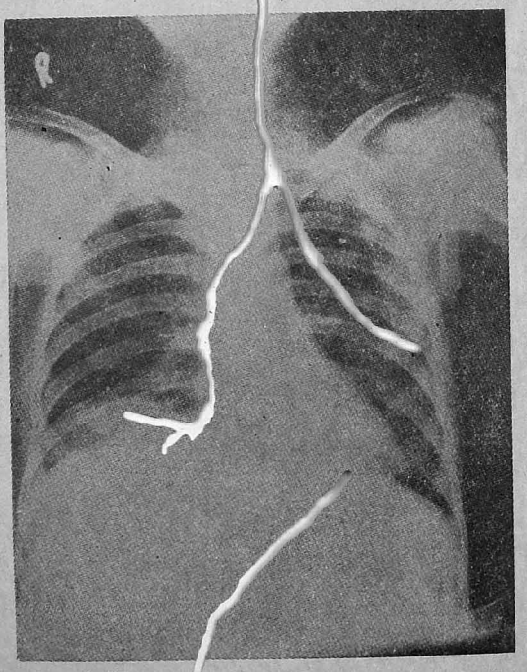

FIG. 4

Same case after treatment (Repeated aspirations, emetine & later chloroquine; Pt. developed broncho pleural fistula & expectorated considerable liver-pus).

FIG. 5

Pleural Biopsy — (Low power): Same case: Showing amoebae (arrow mark) outside muscle layer.

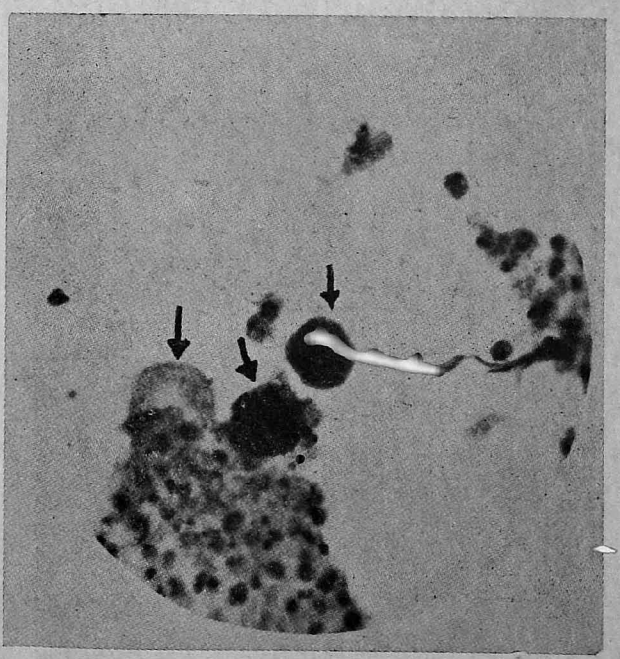

FIG. 6

Same section, high power, showing the amoebae (Iron - Haematoxylin - Preparation).

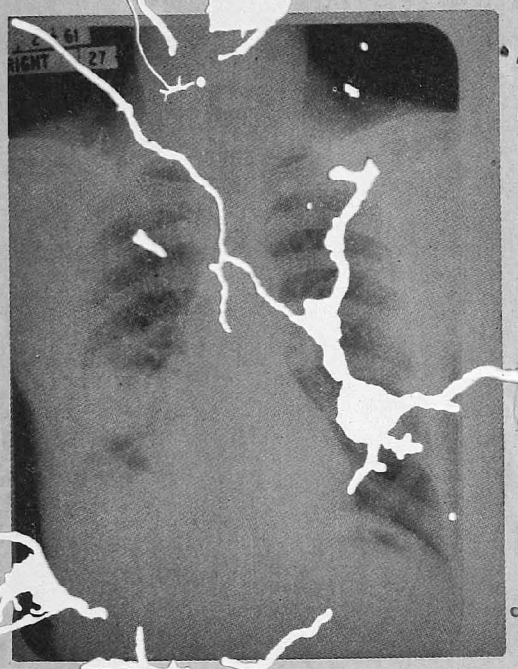

Fig. 7

Secondary Pulmonary Amoebiasis: Rt. dome of the diaphragm - Elevated.

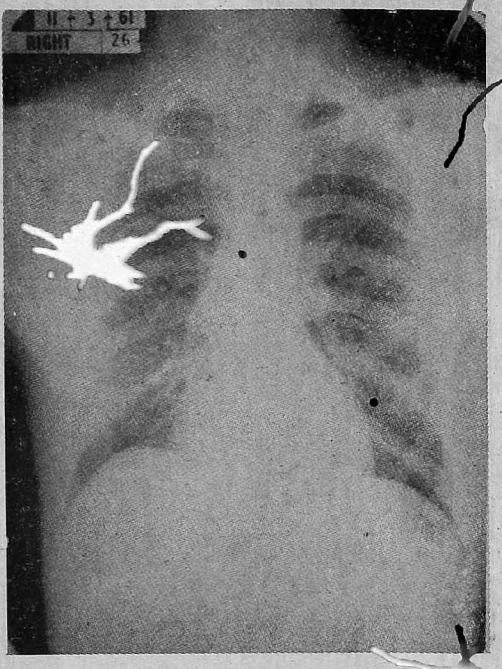

Fig. 8

Same case, after chloroquine.

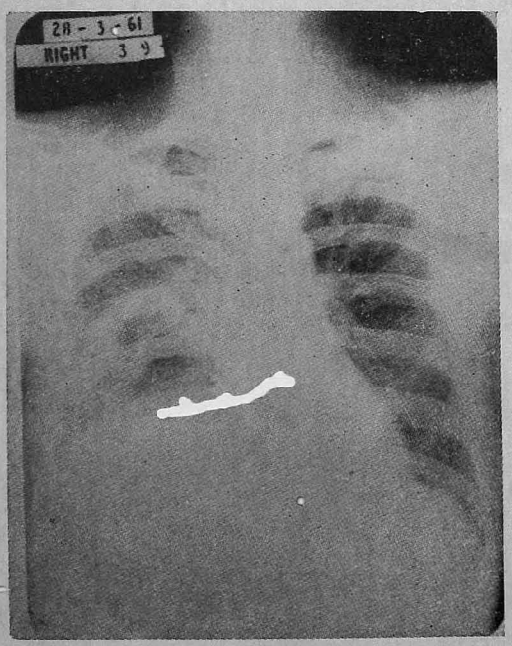

Fig. 9

Secondary Pulmonary Amoebiasis: Abscess in the lung following severe "Pneumonia": Rt. dome elevated. (Pt. had satisfactory resolution following chloroquine therapy).

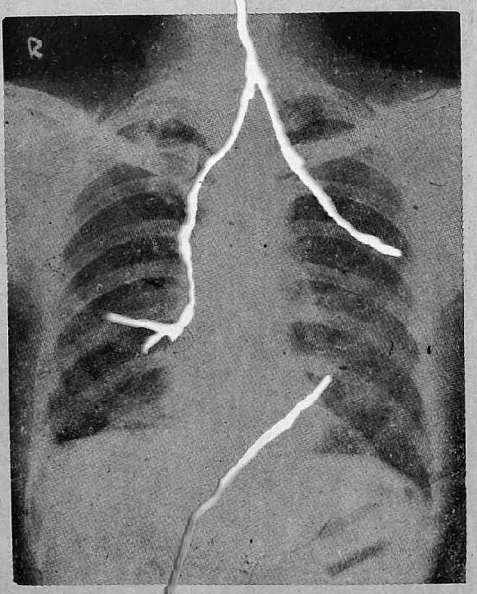

Fig. 10

Suggestive Rt. Basal Shadow of Secondary Pulmonary Amoebiasis.

AMOEBIASIS *

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The recent years have witnessed an increasing interest in amoebiasis. A rapid succession of drugs have been offered for its cure by the pharmaceutical industry, each new drug being claimed to have a lower relapse rate than others already in the field. As yet, we have no single drug that can eliminate both the lumenal (cystic) phase of the parasite and the tissue (vegetative) phase completely.

While formerly there had been an impression that asymptomatic amoebiasis did exist as an entity, its prevalence in the community has been worked out only recently in some studies (Shrivatsava, 1953, Shaw *et al*, 1960) and found to be appreciable, being around 20%; i e, one out of every 5 asymptomatic individuals harbours the parasite. In individuals with a provisional clinical diagnosis made on the basis of the symptoms and signs, amoebiasis is confirmed in 35 to 64% (Shaw *et al*, 1960). It has been suggested by some workers that there has been an increase in the incidence in amoebiasis in recent years (Shaw *et al*, 1960), because of urbanisation, over-crowding, change in eating habits, more people tending to eat in public eating houses, and so on. It is difficult to prove this impression, because data regarding the actual incidence of amoebiasis in the first two decades of this century are meagre and are unlikely to be reliable.

It is interesting to observe that for a disease which has assumed importance in clinical practice, the first recognition of the causative role of Entamoeba histolytica was in 1881 when Koch demonstrated Entamoeba Histolytica as the causative organism of tropical dysentery. Earlier, Lewis and Cunningham and Losch had spotted the parasite but failed to associate with the disease. It is probable that amoebiasis did exist much earlier but as to how ancient a disease it is, it is difficult to assess.

Though predominantly tropical in its distribution, the disease is cosmopolitan and a number of reports have been published to indicate its incidence in temperate zones.

The parasite itself is known to exist in two 'races'. The trophozoites and cysts of the 'small race', also called Entamoeba Hartmanni (cyst size less than 7 microns) are distinctly smaller than those of the 'large race'. It has been held by some workers that the 'small race' is a harmless commensal while the 'large race' is pathogenic (Sapero *et al*, 1942). However, other workers (Meleney and Zuckerman, 1948) have shown that, under suitable conditions, the 'small race' gets transformed into the large ones. Cysts of the 'small race' have been shown to produce lesions in kittens (Frye and Meleney, 1938). Faust (1954) has mentioned

* Based on a lecture delivered before the Nellore Branch of I. M. A. in May 1961.

a case of symptomatic amoebiasis in a child passing cysts of E. H. less than 7 microns in size.

It would, therefore, seem to be unsafe to regard the small race of E. H. as harmless commensals. Even in the large race of E. H., non-virulent strains have been proved to exist. They are morphologically indistinguishable from virulent strains, though it is possible to separate them by animal experiments, the non-virulent, so called carrier strains causing no lesions in rat's caecum whereas virulent strains do. (Neal, 1957)

As for the role of bacteria in the production of the lesions in amoebiasis, it has been shown that the amoeba itself is invasive (Frye and Meleney, 1953; Phillips and Bartgis, 1954) and bacteria which are intrinsically non-pathogenic, like B. Coli, may help in its invasiveness. Pathogenic bacteria aggravate existing amoebic lesions (Neale, 1956). The complete relationship between amoebae and bacteria is not fully understood. However, atleast so far as acute manifestations of intestinal amoebiasis are concerned, drugs which are bactericidal or bacteriostatic have proved to be effective in controlling the symptoms.

While there is no racial susceptibility to amoebiasis, the dietary habits of a community may influence the development of clinical amoebiasis. Elsdon-Dew (1949) has reported on the varying incidence of amoebiasis in the three racial groups, European, Indian and Bantu living in Durban, Natal. Among Europeans, there was only a moderate incidence of the disease, acute amoebic colitis with dysentery being rare; with Indians, infection was widespread but clinical manifestations infrequent; amoebiasis as a disease was widely prevalent and often fulminant in character in Bantus. The relative incidence of the disease in the three communities was probably due to the basically different diets. A diet poor in animal protein and rich in carbohydrates probably predisposes to a high prevalence of the infection. We have, as yet, no satisfactory measure of acquired resistance to amoebiasis. The complement fixation test, which is positive in extraintestinal amoebiasis due to a closer host-tissue and parasite relationship in extra-intestinal situations, becomes negative with the eradication of the infection.

It is not proposed to discuss the familiar pattern of the pathological lesions of amoebiasis. That the Entamoeba Histolytica can remain as a surface-contact parasite in the intestinal wall and, under favourable circumstances, become a tissue invader and produce symptoms is worth observing. This would explain long periods of absence of symptoms after infection has occurred and the varying periods of incubation. In its lumenal phase, it depends for its growth and multiplication on certain bacteria in the intestinal lumen. Once the intestinal wall or other organs of the body are invaded, the host cells appear to provide the stimulus for its activity and the bacteria seem unnecessary. The commonest site for 'Primary Ulceration', the first entry of the amoeba in the intestinal wall are the coecal and the sigmoido-rectal areas. Amoebae emanating in these regions secondarily invade more extensive areas of the large intestines and, rarely, by regurgitation, the terminal ileum.

Though usually, symptoms, vague and of a general nature, may occur in between bouts of diarrhoea or

dysentery in some instances, in the chronic stage of the bowel lesions of amoebiasis, absolutely no relevant bowel symptoms may occur for long periods.

The complications of the bowel lesions include appendicitis (7 to 40% of fatal cases), perforation of the intestinal lesions, both in fulminating acute cases and in the chronic stage, profuse haemorrhage from large ulcers and the development of amoebomas; these are granulomatous lesions presenting as tumour masses most commonly in the caecum, less frequently in the transverse colon and the sigmoid colon.

Extra-intestinal lesions occur most commonly in the liver in which three stages of involvement, of prehepatitis, hepatitis and of a formed abscess are recognised. Recently, it has been shown that the pattern of chronic diffuse hepatitis without abscess formation may occur in hepatic amoebiasis (Doxiades *et al.*, 1961). Though leucocytosis may occur with amoebic hepatitis and with abscess formation, marked polymorphonuclear increase would suggest secondary bacterial infection. Solitary abscesses of the liver are commonest in the right lobe (85 to 95%) and about 50% of them rupture throuh the right dome of the diaphragm.

Pleuro-pulmonary Amoebiasis: The incidence of pleuro-pulmonary complications of amoebiasis has been variously reported as 6·3% (Webster, 1956), 13·5% (Ochsner and DeBakey, 1935), 5·5% (El-Mofti and Mousa, 1951). The pleura and the lung are involved usually secondary to hepatic amoebiasis; consequently, usually the right lower lobe is the site of focal pneumonic consolidation or abscess, due to amoebic invasion. Pleural involvement may take the form of an empyema due to rupture of the liver abscess through the diaphragm or, less commonly as a serofibrinous effusion.

Pulmonary amoebiasis may occur without hepatic amoebiasis-so called primary pulmonary amoebiasis, by spread of the infection from the large intestines via the blood stream (inferior haemorrhoidal veins and to the inferior vena cava), or through the mesenteric lymphatics, ultimately reaching the systemic veins. The liver may thus be completely bypassed.

Lesions in primary pulmonary amoebiasis may occur anywhere in the two lungs, unlike in secondary pulmonary amoebiasis; in the later, by the nature of the origin of the infection from the liver, the lesion is invariably in the right lower lobe. The hepatic abscess may burst into the lung across the thickened basal pleura and be expectorated via the hepato-bronchial fistula.

Amoebiasis of the brain is very uncommon and is mostly due to metastasis from the lungs.

Cutaneous amoebic lesions usually occur at the sites of drainage on to the surface of amoebic abscesses from the liver or from the caecal or colonic lesions. Amoebiasis at other sites as in the spleen and genital organs have been recorded but are extremely rare.

The Clinical Symptomatology of Amoebiasis: Intestinal amoebiasis usually announces itself either with dysentery or diarrhoea. The clinical characters of amoebic dysentery need no elaboration. The comparatively fewer stools than in bacillary dysentery, presence of faecal matter in the stool,

foul smell, acidic reaction and dark red rather than bright red blood in the stool are relevant features. Microscopy reveals the vegetative forms of Entamoeba Histolytica in the stool specimen. A rectal swab and a saline preparation of the material obtained is often more successful in demonstrating the parasite than a direct stool examination. Scanty cellular exudate comprising chiefly of mononuclears and the frequent presence of Trichomonas Hominis are additional features.

Diarrhoea occurring in bouts with intervals of constipation which are more conspicuous, mild colicky lower abdominal pain, excessive or decreased appetite and flatulence may occur; head ache, easy fatiguability, weight loss, depression or irritability are among the general symptoms in amoebiasis. Relief is striking, following proper diagnosis and adequate treatment.

Generally, while vegetative forms are seen in liquid stool samples of dysentery or diarrhoea, cysts are passed in solid or formed stool. It has been worked out that, in examining formed stool for cysts, a single specimen examination would yield 32% and six examinations 72% positive results (Svensson 1955). For practical purposes, three stool smear examinations at 3 to 4 day intervals between them would be advisable and this is more rewarding than 3 consecutive daily examinations (Stamm, 1957). The cysts survive in formed stool, kept in an ice box, up to a week. An iodine preparation should invariably be done when looking for cysts, and a filtered saturated solution of iodine made up to 1% in potassium iodide is more effective than Lugol's iodine itself, which may destroy the cysts.

A specimen of liquid stool, after a saline purge, may reveal the parasite when ordinary stool preparations have been negative. It is better to use sodium sulphate than magnesium sulphate for purgation, since the solution of the latter salt, because of its relative alkaline pH, destroys the the cysts. Concentration methods are not significantly more rewarding than repeated stool smear examinations. Culture method for demonstrating the parasite are only of academic interest. Sigmoidoscopic examination is helpful, when the recto-sigmoid is the seat of the lesion and, obviously, when the lesions are higher up, sigmoidoscopy may yield negative results. Scrapings from the edges of the ulcer or biopsy of the ulcer edge may yield a positive result.

An unusual but important manifestation of intestinal amoebiasis is the socalled appendiceal syndrome. In this the clinical presentation may mimic classical acute appendicitis. It may be impossible to distinguish this manifestation of amoebiasis in the absence of a previous dysenteric history or of attacks of diarrhoea with intervening constipation. Even during the attack of appendicitis, a blood stained stool or loose stool may furnish material for examination and confirmation of the suspicion.

In hepatic amoebiasis, the usual presentation is in the stage of hepatitis with right hypochondrial pain, often brought on by food, stooping, or lying on the right side. Fever and weight loss are inconspicuous. The stage of the formed abscess in the liver may be difficult to distinguish from the hepatitic stage, but persistent fever, weight loss, malaise, and, in well established cases, a muddy discoloration of the skin, localized bulge from the liver surface on clinical or

radiological examination, occurrence of jaundice, failure of the symptoms to subside with antiamoebic therapy-are points of help in distinguishing the advent of abscess formation. In case of doubt, after at least a short course of anti-amoebic therapy, the liver should be explored with a needle.

An example of the amoebic abscess of the liver. which was aspirated is shown in Figs. 1 & 2.

Pleuro-pulmonary amoebiasis is usually of the secondary variety. In this, pleuritic pain over the right side of the chest and pain over the right upper abdominal quadrant and right shoulder, due to associated diaphragmatic pleurisy occurs. Cough may be noticed, with scanty or moderate mucoid sputum, changing to a chocolate coloured expectoration when the liver abscess bursts, producing a hepato-bronchial fistula, or when an empyema containing liver pus bursts into a bronchus, producing a bronchopleural fistula. An example of this latter instance is shown in Figs. 3 and 4, in the case of. an elderly male who had amoebic liver abscess and a secondary empyema which bursts into the bronchial tree. Amoebae were demonstrated in the pleural biopsy material as well as in the empyema fluid (Figs. 5 and 6).

Examples of secondary pulmonary amoebiasis are also illustrated in Figs. 7, 8, 9 and 10. The typical radiological appearance is said to be a triangular shadow over the right lower zone, with the base over the liver and apex towards the hilum (Oschen and De Dakey, 1939).

Primary pulmonary amoebiasis may be difficult to diagnose as the lesions may occur well away from the right dome of the diaphragm; the usual differential diagnosis from other lesions causing solid shadows or cavitary lesions in the lung would have to be considered. Sputum examination for Entamoeba Histolytica may be rewarding in both the types of pulmonary amoebiasis.

Therapy of Amoebiasis: In the treatment of amoebiasis, it is advisable to include in the regimen a combination of two drugs, one against the lumenal phase (cysts) and the other against the tissue phase (vegetative forms) of the parasite. This will ensure that in case colitis is the clinical manifestation, metastatic tissue phase lesions are either prevented or eradicated. Conversely, in treating hepatitis or other metastatic lesions, the primary intestinal lesions are simultaneously treated, though there may be no bowel symptoms.

In communities in which amoebiasis is endemic, and the infestation is heavy and widespread, there is always the possibility of reinfection, and this renders the evaluation of treatment difficult. It is advisable to give a second course of anti-amoebic drugs, a combination of two types of drugs different from those of the first course, after about six to eight weeks, to ensure satisfactory eradication of the infection.

A list of currently available drugs in amoebiasis is long and of a bewildering variety. Drugs against the tissue phase of the parasite are, principally, emetine hydrochloride and chloroquine. Some of the synthetic anti-amoebic drugs, for example phenanthroline compounds (Entobex), biallyl amicol hydrochloride (Camoform), etc., apart from their acclaimed effect on the lumenal phase, and, among antibiotics, Erythromycin, Fumagillin and Paromycin have been

shown to be effective in varying degrees against the tissue phase of the parasite.

Against the lumenal phase of the parasite, emetine bismuth iodide, at least till recently, was known to be most effective, in that it gave the maximum cyst clearance. The side effects of emetine bismuth iodide, particularly nausea and vomiting, within a few hours of administering the drug, are sometimes troublesome. Clinical trials with Diloxanide (Entamide) showed that the drug was among the most effective in the cyst passer with a relapse rate of around 13% (Woodruff *et al*, 1957; Sanjivi and Thiruvengadam, 1958). More recently (Marsden, 1960) even better results were obtained with entamide furoate (Furamide) with a relapse rate of 10% as against 25·3% with emetine bismuth iodide.

To summarise current trends, it may be stated that in acute amoebic dysentery, the treatment would consist of emetine hydrochloride, 1 grain daily intramuscularly, changed over to emetine bismuth iodide when the dysentery is controlled. The course of emetine itself should not exceed a total dose of 10 to 12 grains, and in fact, dysentery is usually controlled by about the fourth or sixth injection of emetine. A course of a drug against the lumenal phase of the parasite like Diloxanide Furoate (0·5 gm. thrice daily for ten days) or emetine bismuth iodide (3 grains daily at bed time after an early light supper, and a dose of 0·75 grain of gardenal for ten days) is given in continuation with emetine therapy. While giving emetine, the usual precautions on account of its cardiotoxic effect must be observed. If the patient's blood pressure is low, or if there is any myocardial disease, instead of emetine, oxy-tetracycline in divided doses of 2 grams per day for 7 to 10 days may be given.

In chronic amoebic colitis satisfactory results are obtained by a preliminary course of penicillin, five lakhs intramuscularly twice daily of the crystalline salt, together with sulpha-thalazole, 6 grams daily in divided doses after which a course of Diloxanide Furoate or emetine bismuth iodide is given. Chloroquine is also added to the Diloxanide or emetine bismuth iodide in a dosage of 0·6 grams of the base for the first two days, and 0·3 gram for the next fourteen days. The latter drug can cause nausea, vomiting, diarrhoea, visual disturbances, vertigo, etc. on the appearance of which the drug has to be temporaily suspended and restarted in smaller doses subsequently.

Amoebic hepatitis and other metastatic amoebic lesions are best treated with a course of chloroquine as mentioned above, in conjunction with a drug against the lumenal (cystic) stage of this parasite. The results with chloroquine in hepatic amoebiasis and other metastatic amoebiasis is as good as with emetine without the possible serious toxic effects of the latter. Wherever abscesses are formed at metastatic sites, prompt aspiration or surgical drainage would be necessary.

My thanks are due to the Director of Medical Services, Madras, and the Dean, Government Stanley Hospital and Stanley Medical College for granting me permission to give the talk and for referring to the clinical material of the hospital; to Dr. K. S. Sanjivi, Honorary Physician, Railway Hospital, Permabur for some of the cases referred to in the text, to all the colleagues in my unit who have helped

me in the clinical study of amoebiasis and to Dr. Smt. V. C. Anguli, Prof. of Pathology, Stanley Medical College, for the pleural biopsy report (Figs. 5 & 6).

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STATE SECRETARY'S ANNOUNCEMENT:

AN APPEAL TO ALL THE MEMBERS

From the 1st of October 1961, the new association year 1961—'62 commences. The strength of the Madras State Branch of I. M. A. as on 30—9—1961 is only 1786. This figure represents but a poor fraction of the total number of medical men in each town of our state who are yet to be brought into our fold. The members will surely agree that in order to strengthen the voice of the association and to attract the attention of the government and other powers, unity in our ranks is most essential. With the co-operation of all our members, a rapid increase in the strength of the Madras State Branch of I. M. A. can easily be accomplished. May I request that each member regards the month of October as the month for a membership campaign and help the Hony. Secretary of his branch to enlist new members? I request the Hony. Secretary of each branch to fix at least an increase of 25% of the membership of his branch as the target, and reach the same during this month.

M. P. JESUDASEN,
Honorary State Secretary,
Madras State Branch, I. M. A.

TUBERCLE BACILLI IN FLOURESCENCE MICROSCOPY

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Introduction: The demonstration of tubercle bacilli under flourescence microscopy is a diagnostic method like the detection of the tubercle bacilli stained by Ziehl Neelson's method for ordinary microscopy. The flourescence microscopy is a relatively inexpensive technique and has many advantages in practice. At present, this method is used in the WHO chemotherapy centre, Madras, and a few other institutions in the Madras State.

In 1904, while experimenting with the ultraviolet dark field microscope, Kohler discovered that some substances emit spontaneous flourescence. Later, in 1907, Kaiserling demonstrated, that the tubercle bacilli has whitish-blue flourescence. But the use of flourescent microscopy was introduced by Hagemann in 1937.

Later, it was described and modifications to simplify the technique by Keller (1938), Kline & Leach (1940), Tanner, Lind & Shaughnessy (1941), Lempert (1944), John Ryley (1945), Norman & Jelks (1945), Clegg & Foster-Carter (1946), G. C. Hughes (1947), Wilson (1952), Von Haebler & Murray (1954), Needham (1957). Recently Else Holst, Michison & Radhakrishna have described the technique employed by them (1954).

Principle of Flourescene Microscopy: Flourescence is a physical character of certain substances, which are stimulated by the absorption of invisible ultraviolet radiation, resulting in the emission of visible light. Though the exact mode of this action is not known, it is presumed that it is due to some electronic derangement. Similar to the ability of retaining the acid-fast dyes, the tubercle bacilli might absorb and retain some flourescent acid-fast materials. This principle gave a new approach to the problem of the detection of tubercle bacilli under flourescence microscopy. This method makes use of the flourescent property of the acid-fast bacilli in ultraviolet light, when stained with Auramine 'O' which is retained by the bacillus after subsequent treatment with acid and alcohol. The acid-fast wax-mycolic acid portion of the bacilli is found to retain carbolfuschin in Ziehl-Neelson's method and auramine in flourescence microscopy staining method.

Equipment and materials: The following material and accessories are required for flourescence microscopy:

1. A standard monocular microscope

2. An electric discharge lamp giving a strong source of ultraviolet light. This may be a multipurpose microscope lamp with mercury vapour bulb. A powerful, concentrated source of light rich in ultraviolet rays may also be obtained with a carbon arc or a 500 watts G. E. projection bulb. The lamp may be mounted in a ventilated sheet metal lamp-box, improvised in the laboratory. The beam of light from the bulb should cover the mirror of the microscope.

3. *A lamp filter:* The concentrated light from the lamp should pass through a blue ultraviolet transmitting filter. A solution of copper sulphate (ammoniated) in a globular

glass flask was previously used as a condenser, filter, and cooler. Now a heat absorbing filter and an exciter filter, BG 12 are fitted to the lamp. The filtered light rays supply power of illumination bright enough to permit the demonstration in a room darkened only by drawing the window shades.

4. *Eye-piece barrier filter:* A mounted minus blue or yellow contrast filter (OG 5) is fitted to the draw tube of the microscope, with a X 10 eye-piece. This filter will absorb the ultraviolet radiation and the blue light transmitted by the lamp and the field appears dark. Further, the filter prevents the ultraviolet rays damaging the eyes of the observer, while viewing the fields under examination.

5. *Objective:* A $\frac{1}{2}$ inch objective is generally used.

6. A diamond objective marker is fitted for marking the doubtful fields of examination, to be stained and confirmed by Ziehl-Neelson's method.

7. *Staining materials:*

(i) Auramine-Phenol Mixture:

Phenol crystals	30 G.
Distilled water	1000 ml.

Warm the solution to 40° C; add auramine 3 G; shake vigorously; filter the liquid; store and use it.

(ii) Acid-Alcohol Mixture:

Sodium chloride	20 G.
Con. hydrochloric acid	20 ml.
Distilled water upto	500 ml.
74·0% alcohol upto	1500 ml.

(iii) Potassium Permanganate Solution:

Pot. permanganate crystals	1 G.
Distilled water upto	1000 ml.

Technique: The technique described by Lempert is applied with slight modifications. A smear of the material to be examined for the presence of tubercle bacilli is made with a slide using a glass rod. The smear is fixed by gentle heat. The auramine-phenol solution is applied on the smear. The slide is not heated, but the stain is allowed to act for about 6 minutes. Then the smear is washed and acidified with a few drops of acid-alcohol solution for 2 minutes. The stain is then flooded off and washed. The smear is counterstained with pot. permanganate solution for 30 seconds.

The slide is kept on the stage of the microscope, the proper relation of the lamp to the condensing lens is determined by trial and error, viewing the appearance of the field in the microscope. The field is viewed first with a low power objective and then with the high power. The auramine stained bacilli on the slide will be stimulated to flouresce by the ultraviolet rays. The bacilli are seen as golden-yellow rods in a dark background.

There will be no necessity to use the oil-immersion lens in routine examinations. But it can be used, if desired. It should be remembered in this connection, that the cedar-wood oil, sandal-wood oil, and mineral oils themselves flouresce and hence unsuitable. The oil of Wintergreen (Methyl Salicylate) is the best material for the oil-immersion lens in flourescent microscopy.

Advantages:

1. In a laboratory, where the number of specimens to be examined is more than 40, this method is advantageous, as it saves time and expense. A full smear can be examined in about 3 minutes with the low power.

2. The chance of detecting the scanty bacilli is greater and it is also easier to distinguish the bacilli, as the field, when using a half-inch objective in flourescent microscopy has approximately 40 times the area available when using an oil-immersion objective in ordinary microscopy.

3. This method is less fatiguing to the examiner, who can easily examine 100 smears in a day.

4. If the bacilli appear atypical and confirmation is required, the same field can be marked on the slide. The same slide can be overstained with the usual Ziehl-Neelson's method and the slide can be examined with ordinary light and oil-immersion microscopy.

5. The tissue sections may also be stained with auramine dye and examined under flourescence microscope.

6. The initial cost of the additional equipment required for the flourescence method is comparatively less. In countries like India, where the culture method cannot be applied widely and the laboratory facilities are limited. the flourescent microscopy can be recommended on economical grounds.

Disadvantages :

1. Under flourescent microscopy, the other elements of the sputum are missed and the morphological character of the bacilli is not visible.

2. If a slide stained with auramine is exposed to ultraviolet rays for about 10 minutes, the staining fades away and fresh smears will have to be prepared again.

3. Though a slide treated with auramine-phenol can be restained by Ziehl-Neelson's method, the Ziehl-Neelson's staining is irreversible and it cannot be restained with auramine-phenol later and it cannot be examined under flourescent microscopy.

Incidentally, it is also intersting to know that the flourescent microscopy technique can also be used to detect the plague bacilli from laboratory cultures and in tissue smears. It can also be used to demonstrate the presence of malignant cells in vaginal secretions and cervical scrapings, which are stained with acridine orange, a flourescent dye.

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NEWS AND NOTES

MADRAS MEDICAL COUNCIL ELECTION

NOTIFICATION.

Drs. G. Srirâmulu, K. Narayanamurthi, C. Nathamuni, D. R. Varman, K. Rama Rao, D. V. Venkappa and V. Vijayaraghavan have been elected as members of the Madras Medical Council from among the registered practitioners of this State for a term of five years on and from the 3rd November 1961. They were the only candidates duly nominated for the vacancies.

G. KRISHNAMURTHI,
Registrar, Madras Medical Council
and Returning Officer.

CONTRIBUTORY HEALTH SERVICE SCHEME

The contributory health service scheme for the central government servants in Delhi and New Delhi is another facet of the social welfare movement based on the principle of joint action by the employer and the employee, to the mutual advantage of both. Prior to the introduction of the scheme, central government servants and members of their families were entitled to free medical aid with many reservations. Under the old system, they had initially to incur the expenditure on their medical treatment under the advice of the authorised medical attendants and get reimbursements of such expenses later on from government to the extent admissible under the rules. This system of reimbursement was a great handicap, especially to the low paid government employees, who could ill afford to incur the initial expenditure in availing of the facilities provided by the government. It had thus been felt for a long time that the medical facilities sought to be given to the government servants as part of the terms and conditions of their service were inadequate in many respects. The contributory health service scheme was designed to remove these defects and to provide a fuller and more efficient and comprehensive medical service to the central government servants and members of their families. It has abolished the distinction between class IV and other classes of government servants in the matter of free medical service and treatment. Under the contributory health service scheme, the lowest employee is entitled to and enjoys the same benefits as the highest officer.

The facilities provided under the scheme include:—

(i) Free medical attendance for the government servants and members of their families at the dispensaries and at their residences. The scope of the term 'family' has been extended to include dependent parents, who were not covered earlier by the civil service medical attendance rules.

(ii) Free specialist consultation and laboratory and x-ray investigation, etc.

(iii) Free treatment to all in hospitals, including free diet to those whose monthly income is less than Rs. 180/-.

(iv) Supply of all medicines that may be prescribed by the medical attendants or specialists, free of cost.

(v) Provision for treatment of dental, ophthalmic and ear, nose and throat diseases by specialists, including free refraction examination.

(vi) Hospitalization and specialist treatment in special hospitals outside Delhi for cases of T. B., cancer, poliomyelitis, including payment of travelling allowance and hospital charges.

	1954	1960
Government servants catered for ...	53,000	not given.
Total population catered for ...	2,23,000	4,56,000
No. of dispensaries		
Static ...	16	39
Mobile ...	...	4
Medical officers		
Assistant surgeons ...	29	195 (sanctioned strength)
Specialists ...	11	36
Attendance at dispensary ...	1,52,927	47,43,968
,, daily average ...	...	15,708
Expenditure Rs.	15,00,000	Rs. 70,00,000
,, Contributions Rs.	7,00,000	Rs. 32,50,000

Facilities provided are:—

(1) Comprehensive medical care

(2) Family planning centres

(3) Check-up clinics

(4) Mass x-ray campaign to detect ' silent ' cases of tuberculosis

(5) Free " Yoga " classes in 6 centres

Contribution from the individual employee varies from 50 nP. to Rs. 12/- a month.

The contributory health service scheme was started as an experimental measure in 1954. The experiment has proved successful and the scheme has served well the purpose of providing comprehensive medical care facilities to the beneficiaries, especially those in the lower income groups. Impressed by its success and its usefulness, the Pay Commission have recommended for its extension to the central government servants in other cities. The extension of the scheme to Bombay, Calcutta and Madras, where the number of central government servants is considerable, has been agreed to and as a first step, it is proposed to be extended to Bombay shortly.

The scheme has thus pointed the way towards medical care facilities being provided on a contributory basis. Its success has led to a number of other agencies and organisations making efforts to bring into being similar schemes on a smaller scale and it is hoped that some day the scheme will prove to be the foundation for a National Health Insurance Service by covering the whole of the country.

LEDERLE MEDICAL RESEARCH FELLOWSHIPS

NEW YORK, N. Y. — Twenty-five outstanding young physicians from all corners of the world will study in the United States this year as participants in the 1961—62 Lederle International Fellowship program.

Since its inception in 1954, the Lederle Fellowship program has assisted 150 foreign physicians in furthering their medical educations. To date, almost a million dollars have been invested in the program by its sponsor, Cyanamid International's Lederle Laboratories. Approximately $ 125,000 has been allocated for this year's activities.

The purpose of the Lederle Fellowships is to support post-graduate study and research by foreign doctors in the United States. According to Dr. F. C. Ottati, Director of Medical Research for Cyanamid International, "We feel that the exchange of medical knowledge and research techniques among physicians, which is made possible through these grants, is a significant contribution to medical practice throughout the world."

The Lederle grants, provide living expenses for one year while the recipient is in the United States, as well as defraying the cost of travel from the doctor's home to his place of study in the U. S.

The fellowships are awarded to a young physician who, in the opinion of top medical authorities in his country, shows exceptional capabilities and promise in his speciality. He is permitted to carry out his advanced studies or research at the school or institution of his choice.

The sponsors who recommend the grants are top-ranking physicians from medical research institutes, deans and faculty members of medical schools or directors of world renowned hospitals.

To date, the awards have been made to physicians from 43 countries and Puerto Rico.

ABSTRACTS AND EXCERPTS

PROTEIN MALNUTRITION IN YOUNG CHILDREN :

Treatment : The basic treatment of kwashiorkor is dietary. The child should be given a diet providing 3 to 5 grams of proteins of good biological value per kilogram of body weight per day. Cow's milk in any form is a convenient and effective source of protien. However, good results can be obtained with adequate combinations of proteins of vegetable origin and even with mixtures of amino acids. Other foods should be progressively added so that by the second or third week of treatment the child is receiving a complete and varied diet, including fruits, vegetables, eggs, meat, and cereals, as well as milk.

Caloric intake should be high enough to insure good protein utilization. After the first few days of adaptation to the diet, an intake of about 150 calories per kilogram of body weight, together with adequate protein, gives good results in most patients with kwashiorkor. In cases of marasmus or in very marasmatic cases of kwashiorkor, a higher intake of calories may be necessary after the initial phase of recovery.

It has been repeatedly demonstrated that, with good dietary treatment, there is no need for additional vitamins or minerals in most cases. Only when clinical or biochemical evidence of a severe deficiency of a specific nutrient is observed, is it desirable to administer a vitamin or other specific nutrient. Children with megaloblastic or iron-deficiency anemias or ocular signs of vitamin A deficiency are among those requiring specific treatment.

Immediately after admission to the hospital and before initiation of the dietary treatment, measures should be taken to correct electrolyte imbalance in these children, particularly if clinical dehydration is present. Particular attention should be given to correcting potassium deficiency.

An antibiotic or a sulfonamide, or both, should also be given during the first few days of hospitalization, even if the child does not give evidence of infection. Severe infections, particularly bronchopneumonia, may develop with few clinical manifestations and be responsible for unexpected deaths during the first few days after admission. Obviously, if infection is found at any stage, it should be treated promptly and effectively, since it interferes with adequate recovery.

It has also been found that good nursing and general care in the hospital, including adequate control of intake, isolation from infectious cases, cleanliness, frequent changes in position during the period of apathy, and affectionate attention at all times, are extremely important for good recovery.

Prevention: Even when good food is consumed, several preventable conditions may interfere with its utilization. Heavy infestations of ascaris and other intestinal parasites, as well as frequent diarrhœa of infectious origin, require specific therapy, and improved environmental sanitation is necessary to prevent their occurrence. Improved sanitation thus becomes a major factor in the prevention of kwashiorkor, along with immunization against smallpox, diphtheria, whooping cough, and tetanus, and also against measles. Prompt and effective therapy for acute infections, both intestinal and systemic, will, of course, help to limit their adverse effects on nutritional status.

— *Scrimshaw, N. S. & Behar, M. Science, 133,* **2045** *(1961).*

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ROUTINE IMMUNIZATION WITH ORALLY ADMINISTERED ATTENUATED POLIOVIRUS. A STUDY OF 850 CHILDREN IN AN AMERICAN CITY.

(J. S. Pagano, S. A. Plotkin, C. C. Janowsky, S. M. Richardson and H. Koprowski. Journal of the American Medical Association 1889, August 27, 1960).

Recent epidemics of poliomyelitis in the United States have occurred chiefly in the crowded poorer sections of cities and affected particularly children under 5 years of age. The need for new methods of immunization, especially in infants under 6 months, prompted the Department of Bublic Health, Philadelphia, in the spring of 1959 to examine the feasibility of early immunization of normal children aged 6 weeks to 6 years (mostly Negroes), living in two low-income districts of the city. The children, none of whom had received Salk vaccine, were given monovalent vaccines by mouth at monthly intervals in the following order: Chat (Type 1), W-Fox (Type 3), and then P-712 (Type-2). Altogether 850 Type-1, 805 Type 3, and 335 Type 2 vaccines were given in milk or cocoa. All three types of vaccine were given to 335 children out of a total of 850; over one-half of the children in the series were under 6 months of age.

Serological sampling revealed that 44% of all the children and 66% of those 4 months to 3 years old were without any poliomyelitis antibodies before vaccination; in virtually all children between 6 and 12 months of age, the antibody titre had fallen to non-detectable levels. Although there was one death, that of a 3 month old child, due to an unexplained cause, no illness that could be attributed to the vaccination was encountered. A significant antibody response was noted in 91 to 100% of those children who had not yet lost maternal antibodies, while in 84 to 100% of those more than 6 months old who had no antibodies before vaccination, a fourfold rise in titre occurred. The proportion of children with antibodies to all three types of virus increased from 19 to 85% after vaccination. The authors state that none of the 22 cases of poliomyelitis in Philadelphia in 1959 occurred in vaccinated children or in their households.

— *Abstracts af World Medicine, March 1961, Vol. 29 page 140.*

TREATMENT OF CHRONIC FURUNCULOSIS:

(L. G. Tulloch, V. G. Alder, and W. A. Gillespie. - British Medical Journal - 354–356, July 30, 1960).

The authors of this paper from the United Bristol Hospitals describe the treatment of 58 adult patients suffering from recurrent furunculosis of at least 6 months' duration. Alternate patients were placed in either a treatment group or a control group and from all the patients swabs were taken at monthly intervals from a furuncle, the anterior nares, the ears and eyelids, and later in the trial from the perineum; swabs were also taken from the relatives of some of the patients. In both groups of patients the boil-bearing areas were swabbed with a 1 in 3,000 solution of mercuric chloride twice a day.

The treatment group, but not the controls, were given an antiseptic cream, usually consisting of a combination of neomycin and bacitracin, to apply to the anterior nares 2 or 3 times daily for 3 months. The same cream was also applied to the eyelids of those patients in whom culture of the eye swabs was positive in the absence of any clinical evidence of inflammation; if obvious blepharitis was present, a cream containing hydrocortisone and neomycin was used. Ear-drops with these active ingredients were used in the treatment of otitis externa, while a hexachlorophane bath or a talcum powder containing 3% hexachlorophane was prescribed for patients in whom cultures of perineal swabs were positive.

In all, 33 patients in the treated group and 23 controls were followed up for 4 to 6 months. Of the controls, 3 were cured and 20 continued to have boils; of the treatment group, 22 remained free from lesions, one improved, and 5 were unchanged. In the remaining 5 cases, family source of re-infection had to be eradicated before cure was effected. Cultures taken from the anterior nares remained positive in 5 out of 24 patients in the treated group and in 17 out of 20 controls. Nasal swabs were taken from 22 families, and from 14, strains which were similar to those in the patients were isolated from one or more relatives. All except 7 of the 58 patients were nasal carriers of the strains which caused the boils.

The authors emphasize the importance of obtaining the patients' co-operation in the successful treatment of the staphylococcal carrier state.

— Abstracts of World Medicine, March 1961 - Vol. 29 - page 174.

* * * *

HOSPITAL INFECTION YESTERDAY AND TODAY

Dr. Mary Barber of Postgraduate Medical School of London, introducing a symposium on hospital infections writes in the Journal of Clinical Pathology (January 1961—p. 9).

"As pointed out by Lister, hospital infection is a preventable disease. That it has not yet been prevented is largely a reflection on the fact that in the past we have underestimated the enemy. If we are to sueeeed we must

attack the problem on many fronts. Different methods of attack and their effects are described in detail in the subsequent articles of this series. Here it is only possible to outline the strategy".

"The first line of attack must be on the primary human reservoirs of infection. Infected patients must be excluded from open general wards and the infection controlled, where possible, by means of intelligent chemotherapy. With staphylococci, nasal and skin carriers have also got to be considered. To what extent this would remain necessary once the cycle of cross infection had been broken by the strict isolation of infected patients remains to be determined. But at present there is no doubt that carriers, at any rate of multiple resistant staphylococci, can be a source of danger".

"*Staph. pyogenes*, *Ps. pyocyanea*, and other drug resistant coliform bacilli in the hospital environment must also be dealt with. This entails rigorous aseptic technique in wards as well as in theatres, clean air, clean bedding, and hygienic methods of dust removal".

"Last, but not least, some control of the use of antibiotics in hospitals is essential both to prevent the emergence of drug-resistant bacteria and also to avoid rendering patients more susceptible to infection by such bacteria, through elimination of the normal flora".

"Cross-infection in hospital has remained a problem yesterday and to-day. If enough people take it seriously, there is no reason why it should continue tomorrow".

An old lady who was looking forward to the near approach of her hundredth birthday had the misfortune to have a fall in her house, and to bruise—but only slightly—her face on the carpet. The family sent off at once for the family doctor, who was greeted by the old lady asking "Doctor, shall I be disfigured for life?"

* * * *

An asylum inmate had been complaining about a cat in his stomach, tearing around and clawing him. One day he got an attack of appendicitis and the surgeon decided this was a good time to effect a cure. He sent for a black cat and when the inmate came out of the anaesthesia, the surgeon held up the animal and said, "You're okay now. Look what we removed from your stomach."

The inmate scared, yelled out, "you got the wrong cat. The one I swallowed was grey!"

LETTER TO THE EDITOR :

SEMI DOCTORS

It is painfully shocking to see in the press and elsewhere under correspondence the views expressed as regards the deliberations about the recent Hyderabad session of the conference of the health ministers of states and the remedy adopted by them to provide medical relief to our people owing to shortage of doctors A more unwise step and a deleterious one cannot be thought of in the profession of medicine. It speaks of the colossal ignorance of the living conditions and the state of affairs in our country. My attention was drawn about the recent opening of medical schools again by the Mysore Government which I had termed as a retrograde step. On the top of it the creation of 'semi doctors', as they are called in our state, is a paradox un-understandable and unimaginable. That the health ministers of states should have conceived this dangerous step of giving a short training to young men and young women ostensibly on the score of medical relief are rendering a disservice not only to the people of our country, but to the entire profession of medicine. All of us are aware that a little knowledge is a dangerous thing and much more so in the realm of medical science which has advanced by leaps and bounds and require a highly qualified personnel for admission into Medical Colleges to undergo the strenuous courses and become doctors. Therefore, these short-trained young men and young women even to be trusted to treat ordinary ailments are a source of danger to society, as they would produce more complications which could be nipped in the bud by efficient treatment.

The Madras state has been a forward state and had gone ahead of other states in the matter of medical reforms such as a uniform standard of medical education, unification of the civil medical services, etc. Ministers of health in this state in the past have been responsible for these reforms in conjunction with the leaders of the profession and medical associations. What surpasses now is, I consider, a tragedy and if persisted more and more harm would be done to the public. As custodians of the health of our country, state health ministers should have a broader vision and outlook in matters of public health and medical education and not embark on schemes which are doubtful and dangerous. There is no dearth of medical men in our state for medical relief provided the requisite emoluments and ameneties are afforded and particularly so in rural areas. I implore on the ministers of health in different states to give up this dangerous step which would be only augmenting the quackery already rampant in our country. Lastly, I consider the attempt on the part of the state ministers as a challenge and an eye opener to the profession to stick up to its ideals of rendering relief to human suffering irrespective of any reward and thus maintain the dignity, integrity and honour of the noble profession of medicine in all spheres.

The exploration of indigenous medicine is still in the making and no practical and forward steps are taken in affording the needed research. The fact that the L. I. Ms. and G. C. I. Ms. have taken to modern scientific medicine after their integrated course is proof positive of the popularity of modern medicine which they practise.

D. V. VENKAPPA,
Ex-President, I. M.A., A. I. M. L. A., & Member, Medical Council of India.

ASSOCIATION NOTES

BRANCH NOTES

Erode Branch:

A meeting of the Indian Medical Association, Erode branch was held on 26—8—1961, Dr. (Capt.) R. S. K. Raman presided. Dr. S. Subramaniam, M. B., B. S., Chief Medical Officer, Government Hospital, Erode, spoke on the "Upgrading of the present Government Hospital, Erode. In the course of his talk, Dr. Subramaniam made out the following points:—

1. With some alterations, the present hospital be made to accommodate 2 wards of 28 patients, thus bringing up the bed-strength to 156, without incurring any extra cost. Besides, in the first fioor 2 wards could be built, with the result we can have an hospital of 212 beds catering to the needs of Erode and the surrounding villages.

2. A blood bank is an absolute necessity in Erode, with a population of nearly a lakh and where major surgery is being done.

3. The x-ray department has to be made up to date with facilities for screening and fitted with physio-therapy apparatus.

4. An up to date and well-equipped laboratory is a dire necessity for proper diagnosis.

5. At present it is proposed to allot 6 beds to patients suffering from T. B. The leprotic patients are being examined and treated once a week by visiting doctors. Their visits will be increased to 2 per week.

After the talk, various suggestions for improving the present hospital were made by doctors present. A resolution requesting the Government to start a blood-bank was passed unanimously.

Dr. P. K. Rajan was the host for the meeting.

Madurai Branch:

A monthly meeting of the Madura Medical Association was held on Saturday the 9th September, 1961, under the presidentship of Dr. Abdul Sattar, L. O., Madurai. The following condolence resolution on the sudden demise of Dr. K. G. Ramabadran, L. M. P.. Vice-President and life member of our association was passed. "This meeting of the Madura Medical Association unanimously places on record its deep sense of sorrow at the demise of Dr. K. G. Ramabadran, the Vice-President of our Association. The Secretary is requested to convey our condolences to the bereaved family". Dr. R. P. Dhanda, M. S., D. O., M. G. M. Medical College, Indore., gave an interesting lecture on "Electro-Retinography and Vitamin A Deficiencies," and Dr. M. K. Krishna Menon, B. A., M. D., Director, Institute of Obstetrics

and Gynaecology, Women & Children Hospital, Madras, gave an interesting lecture on "Obstetric Emergencies in General Practice". The meeting terminated after an interesting film show on "Sleep & Hypnosis" and Five Year Plan.

Nagapattinam Branch:

A meeting of the I. M. A. Nagapattinam was held at the Government Hospital, Nagapattinam presided by Dr. A. Thiagarajam, M. B., B. S., on 6—8—1961.

A condolence resolution was passed at the death of Dr. U. Krishna Rau, Speaker of the Madras Legislative Assembly, all the members standing in silence for a minute.

Tiruchy Branch:

A monthly meeting of the association was held an Saturday, the 26th August, 1961 in the Association premises, Tiruchy. Dr. P. V. Sundaram, the President was in the chair. The following condolence resolutions were moved from the chair and passed unanimously all members standing in silence for 2 minutes.

"This meeting of the Tiruchy Branch of I. M. A. records with great regret the sad demise of Dr. U. Krishna Rau, M. B., B. S., M. L. A., Madras".

"This meeting of the Tiruchy Branch of I. M. A. records with great regret the sad and sudden demise of Mrs. Dharmambal, wife of Dr. P. E. Jambunathan".

A monthly meeting of the Association was held on Saturday, the 16th September, 1961 in the Medical Association premises, Tiruchy. 38 members were present. Dr. P. V. Sundaram, the President was in the chair.

A resolution congratulating Dr. T. V. Srinivasan on his election as the senior vice-president for the Madras State Branch of I. M. A. for the year 1961—1962 was moved from the chair and passed unanimously.

Dr. P. K. Kalyanaraman, M. B., B. S., Hony. Physician, Government Head-quarters Hospital, Coimbatore give a talk on "Myocardial Infarction and the General Practitioner". The lecturer gave an excellent talk on the subject which was followed by a lengthy discussion.

ERRATA

In the case report on "A Case of Massive Pneumonia due to Friedlander's Bacillus Infection" published in pp. 88—92 in our September, 1961 issue Vol. xxviii No. 3, the author's name should read as "N. Ramachandran" instead of "N. Chandrasekaran". The error is regretted.

MANAGING EDITOR.