ovisacs but were most common in the younger adult females. The distribution of the infections according to the stage of development of the cyclops, as found among 1,302 mixed specimens of *M. leuckarti* and *M. hyalinus*, is given in table II.

TABLE II

Distribution of natural cyclops infections according to stage of development of the cyclops

Life stage of cyclops	Number examined	Number infected	Per cent
Young adult females	522	23	4.4
Immature males and females.	618	18	2.9
Mature males ..	122	4	3.2
Mature females with ovisacs.	40	0	0.0
All forms ..	1,302	45	3.7

As far as could be determined, the guinea-worm larvæ went through the same developmental changes in the body cavities of naturally-infected cyclops as they do in the case of laboratory infections. The possibility of transmission of dracontiasis by these naturally-infected cyclops was demonstrated by the finding at autopsy in dog no. 28 of 51 immature guinea-worms (Moorthy and Sweet, 1936). This dog was fed with 88 naturally-infected cyclops and died 67 days later.

Summary

After repeated unsuccessful attempts made in earlier years after the first of March, cyclops naturally infected by guinea-worm larvæ were found in a step-well in the Chitaldrug district of Mysore State in January and February 1936, at a time when there were in the village several cases of dracontiasis in the early stages. Although *M. leuckarti* had the higher infection rate, the actual number of *M. hyalinus* infected was greater; this was due to its numerical preponderance. No infections were found in other species of cyclops and none of the infected specimens were fully-mature females bearing ovisacs; all infections were in the younger forms of both sexes. That the naturally-infected cyclops could transmit dracontiasis was demonstrated by the finding of immature guinea-worms at autopsy of a dog which died 67 days after a feeding of 88 naturally-infected cyclops.

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PELLAGRA IN VIZAGAPATAM

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and

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PELLAGRA is not an uncommon disease in the southern presidency. Lowe reported 40 cases in Dichpalli, Hyderabad (Deccan), in 1931 and Raman reported 4 cases in Guntur in July 1933; the first case observed by him was in September 1930.

The first case seen by the senior author in Vizagapatam was in January 1933, and, subsequent to that, every year one or two cases have been admitted to the King George Hospital with typical signs and symptoms of pellagra. Short notes of some of the cases are given below :

Case 1.—K. R., aged 40 years, Hindu male. Admitted in King George Hospital on the 31st January, 1933, and discharged on the 25th February. The patient was admitted with general anasarca, pleural effusion on the right side, and pigmented patches of the skin over the dorsum of both feet, the extensor aspects of both legs and thighs, and the extensor aspects of the forearms and lower third of the arms, and a circular ring of pigmentation round the neck. His tongue was glossy and atrophic. The nervous system was normal. Plasma proteins were 6.14 per cent, of which albumin was 0.9 per cent, globulin 5 per cent, and fibrinogen 0.2 per cent; blood urea was 29.6 mg. and blood cholesterol 114 mg. Urine was normal.

Case 2.—M., aged 40 years, Mohammedan male convict, seen in April 1933. Symmetrical pigmented patches over the skin of the forearms, on the lower half of the arms, chiefly on the extensor aspects, and on the dorsum of both hands. There were similar patches on the dorsum of both feet, and anterior and extensor aspects of the lower third of both legs, and there was a patch on the posterior aspect of the neck on the right side. Tongue showed glossitis with atrophy. Nervous system was normal. There was a scar on the abdominal wall (gastro-jejunostomy). No further investigations were done in this case.

Case 3.—A., aged 25 years, Hindu male. Pigmented patches over the dorsum of both hands, on the extensor aspects of both forearms and lower third of arms. Similar patches on the dorsum of the feet and extensor aspects of both the legs. Tongue showed slight glossitis and no atrophy. There was diarrhœa present, patient having about 10 to 12 loose motions a day. There were marked mental symptoms, periods of mental dullness alternating with acute maniacal symptoms. Patient was dirty in his habits. No investigations could be done in this case as the relations took the patient away a short time after admission.

Case 4.—D. A., aged 32 years, Hindu male. Admitted on 3rd October, 1933, and discharged on the 11th November. Pigmented patches on the dorsum of both wrists and feet; a marked pigmented patch round the base of the neck; tongue atrophic and showed extensive glossitis; diarrhœa was present. Other systems—normal.

Blood counts: Blood smear showed nothing abnormal. Red cells—3.86 millions, white cells—6,956, hæmoglobin—60 per cent. Differential count: polymorphonuclears—60.5 per cent, lymphocytes—29.1 per cent, mononuclears—7 per cent, eosinophils—3.5 per cent. Van den Bergh reaction: direct delayed—positive, faint; indirect—positive, faint. Urine—normal. Wassermann reaction—negative. Gastric analysis—normal.

Case 5.—V., aged 20 years. Hindu female (plate V, figure 1). Admitted into Dr. Kutumbiah's wards of the

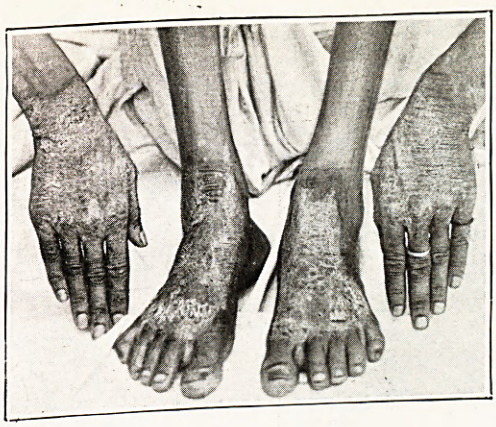

Fig. 1.

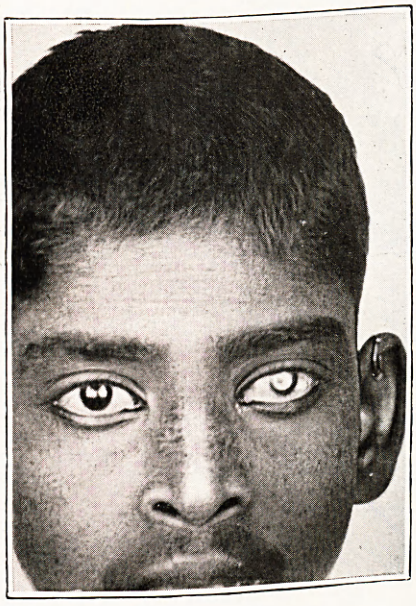

Fig. 2.

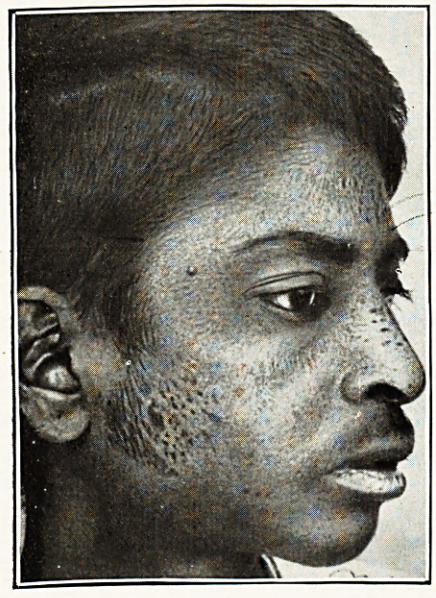

Fig. 3.

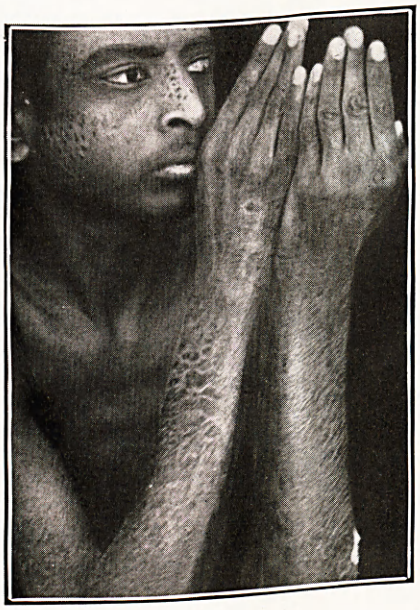

Fig. 4.

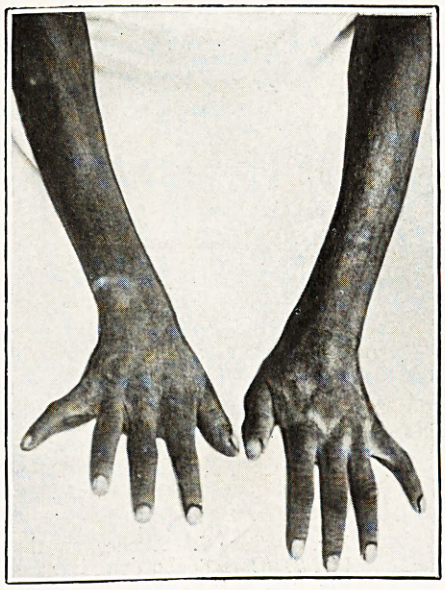

Fig. 5.

Fig. 1.—Case 5 showing the symmetrical patches on the dorsum of both the hands and feet.
Fig. 2.—Case 6. Patch on the nose.
Fig. 3.—Case 6. Patch on the forehead and the nose and on the cheeks (symmetrical).
Fig. 4.—Case 6 showing patches on face and both the hands (symmetrical).
Fig. 5.—Case 7. Patches on the dorsum of both the hands (symmetrical).

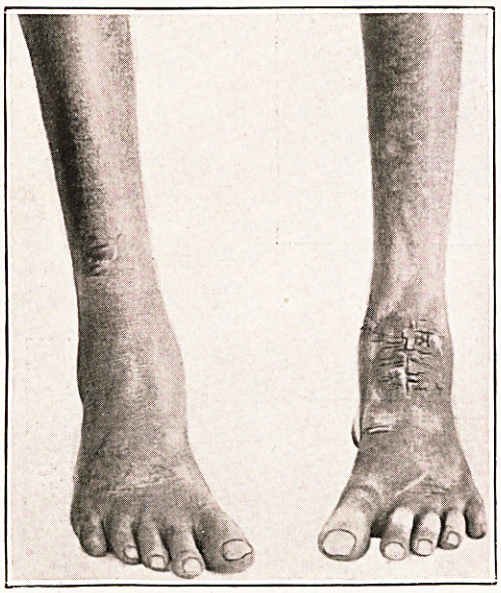

Fig. 6.

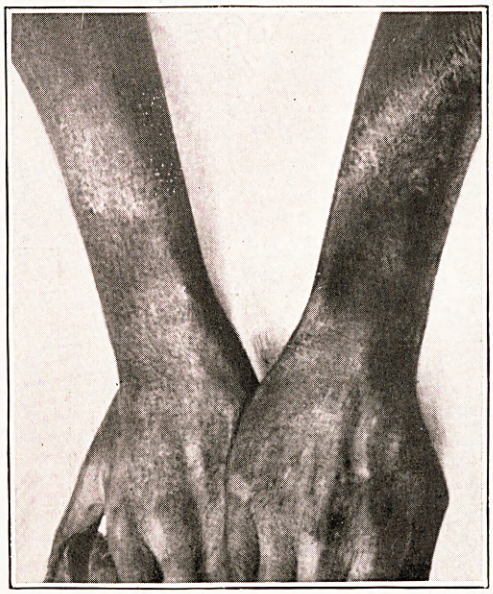

Fig. 7.

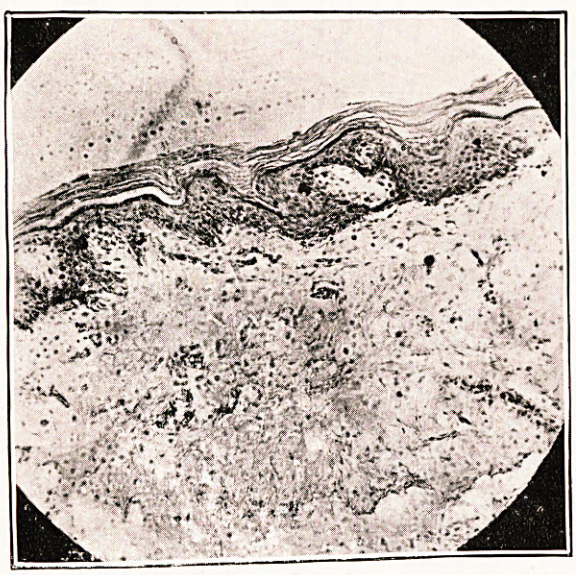

Fig. 8.

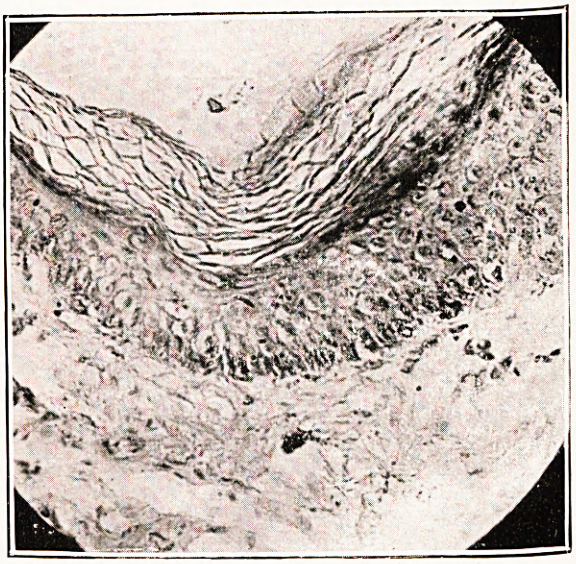

Fig. 9.

Fig. 6.—Case 7. Patches on the dorsum of both the feet (symmetrical).
Fig. 7.—Case 8 showing the rash on dorsum of both the hands.
Fig. 8.—Photomicrograph of the skin (low power).
Fig. 9.—Photomicrograph of the skin (high power).

King George Hospital on the 8th July, 1934. Well-marked pigmented patches on the dorsum of both hands and feet; subjective sensations of heat in the affected areas, anæmia, well-marked glossitis, atrophy of the tongue and mental dullness, no diarrhœa. Other systems—normal. Urine—normal.

Blood: Red cells—1.78 millions, white cells—8,000, hæmoglobin—70 per cent. Differential count: polymorphonuclears—70 per cent, mononuclears—8 per cent, lymphocytes—20 per cent, eosinophils—2 per cent. Blood plasma proteins—5.87 per cent, of which albumin was 2.98 per cent and globulin 2.58 per cent. Calcium—9.46 mg., phosphates—3.46 mg., urea—20 mg.

Case 6.—D. S., aged 20 years. Hindu male (plate V, figures 2, 3 and 4). Admitted on the 3rd December, 1935, for diarrhœa. Tongue atrophic with glossitis; liver slightly enlarged; spleen slightly enlarged. Other systems—normal. Well-marked pigmented patches on the dorsum of the feet and hands, extensor aspects of forearms, on either cheek and over the base of nose. A few patches are seen on the ears.

Blood: Red cells—3.4 millions, white cells—13,750, hæmoglobin—58 per cent. Blood showed microcytic anæmia. Differential count: polymorphonuclears—60 per cent, lymphocytes—30 per cent, mononuclears—1 per cent, eosinophils—9 per cent. Van den Bergh reaction: direct—positive delayed, faint; indirect—positive, faint. Fragility of red cells—hæmolysis began at 0.5 per cent saline and was complete in 0.25 per cent saline. Fæces contained no amœbæ. Blood total proteins—2.03 per cent, albumin—0.94 per cent, globulin—0.89 per cent, gastric function—hypochlorhydria, urine—normal, blood urea—43.1 mg., uric acid—3.49 mg., calcium—10.6 mg., phosphates—3.42 mg., blood sugar—80 mg. per 100 c.c.

Section of skin (plate VI, figures 8 and 9) shows well-marked keratinization of the superficial layers, absence of glossy layer, deficiency of pigment in the pigment-bearing layer, absence of sebaceous glands, and degenerative changes in the hair roots. The dermis shows moderate hyaline fibrosis and no inflammatory infiltration.

A second test meal done on the 20th January, 1936, showed normal gastric acidity.

Case 7.—K., aged 20 years, Hindu male (plate V, figure 5, and plate VI, figure 6). Admitted on 5th December, 1935, for tingling and numbness in the extremities and diarrhœa; slight œdema of the lower extremities; well-marked pigmented patches on the dorsum of the hands and feet and a ring of pigmentation round the neck (Cassel's necklace); anæmia, atrophy of the tongue, and glossitis. Nervous system showed deep reflexes exaggerated, but Babinski was not present. Blood smear shows slight anisocytosis and poikilocytosis. Blood picture—3.86 millions, white cells—7,500, hæmoglobin—60 per cent. Differential count: polymorphonuclears—47 per cent, lymphocytes—45 per cent, eosinophils—7 per cent, mononuclears—5 per cent. Blood Wassermann reaction—doubtful. Gastric function—normal.

Blood urea—50 mg., uric acid—3.6 mg., calcium—9 mg., phosphates—2.89 mg., and sugar—89.7 mg., per 100 c.c. Urine—normal.

Section of skin: Changes are similar to those seen in section of case 6 but the atrophy of the epithelial papillæ is more marked in this case.

Van den Bergh reaction: direct—negative; indirect—positive, faint. Fragility of red cells—hæmolysis begins at 0.5 per cent saline and complete in 0.25 per cent saline.

Case 8.—J. S., aged 40 years (plate VI, figure 7). Admitted on 15th March, 1936. Well-marked pigmented patches on the dorsum of both hands and feet and the extensor aspects of both legs and forearms. The patient complained of indigestion but there was no diarrhœa; no atrophy of the tongue; other systems normal.

Blood picture: Blood smear shows slight anisocytosis and poikilocytosis. Red cells—3 millions, white cells—10,000, hæmoglobin—70 per cent. Differential count: polymorphonuclears—66 per cent, lymphocytes—18 per cent, mononuclears—7.5 per cent, eosinophils—8.5 per cent. Van den Bergh reaction: direct and indirect—negative. Fragility of red cells—hæmolysis begins at 0.5 per cent saline and is complete in 0.35 per cent saline. Gastric analysis showed nothing abnormal except hypermotility. Serum proteins—5.1 per cent, of which albumin is 3 per cent and globulin is 1.83 per cent. Calcium—11.94 mg., phosphates—5 mg., blood urea—20.2 mg., per 100 c.c. Wassermann reaction—strongly positive.

The chief clinical features of all these cases correspond to the descriptions of pellagra given in the various textbooks. It is only a minority that showed any involvement of the central nervous system. The most characteristic feature of the disease is the pigmentation of the skin. The rash usually appears on the extensor aspects of both the hands and the feet and is symmetrical. The pigmented patches are dry and scaly. The margins are well defined. When the scales are peeled off, they leave a surface paler than normal skin. The appearance of the rash on the extensor aspects, its symmetrical distribution and its circumscribed nature is characteristic of pellagra.

The blood proteins were estimated in four cases. The average plasma protein was 4.8 per cent, of which albumin was 1.95 per cent and globulin 2.58 per cent. Blood cholesterol was not appreciably altered in the only case in which it was investigated. Similarly calcium, phosphates, glucose and urea were within normal limits in the cases in which they were estimated.

Case 5 showed anæmia of the hyperchromic type while cases 6 and 7 showed anæmia of the hypochromic type. Van den Bergh test was done in 4 cases and showed no particular abnormality, except for direct delayed positive faint and indirect faint positive reaction.

Gastric analysis was normal in two cases, while there was hypochlorhydria in case 6, which subsequently became normal.

The fæces contained no parasites in cases where they were microscopically examined.

From this analysis the only abnormality that was more or less consistently found in these cases was a diminution in the plasma proteins particularly of the albumin fraction, with an alteration in their relative proportions.

The causation of pellagra is still a matter of controversy. The chief views held are: that it is due to a dietetic deficiency of biological proteins; that it is due to a deficiency of vitamin G or B_2; and the more recently formulated theory of Castle that pellagra along with sprue and pernicious anæmia is due to a deficiency of the anti-anæmic factor.

The persistent deficiency in the plasma proteins in the cases investigated by us suggests a defect in the protein intake particularly meat, eggs and milk. There is in addition also a certain amount of mineral deficiency as shown by the presence of hypochromic anæmia in two

cases and of the anti-anæmic factor in one case*.

Treatment.—Case 1 was entirely treated with diet which contained milk and eggs and the patient was discharged cured. Cases 2 and 3 were not treated in hospital. Case 4 was treated with a diet rich in proteins and daily injections of a 10 per cent solution of sodium thiosulphate in distilled water intravenously starting with a dose of 5 c.cm. and rising to 10 c.cm. This patient was discharged cured but was admitted again the next year with recurrence of the symptoms. Case 5 was treated with high protein diet and sodium thiosulphate injections but the patient requested to be discharged before any improvement could be noticed. Cases 6 and 7 were treated with diet rich in proteins, sodium thiosulphate injections and liver soup. It is in these two cases that the recovery was most rapid. Case 8 was treated only with a protein-rich diet and liver but without sodium thiosulphate injections. In this case also the recovery was equally rapid and the patient left the hospital cured.

We cannot say how far sodium thiosulphate was responsible in the cure of cases 4, 6 and 7. In all probability, its value was slight as in case 8 recovery was even better without it. The recovery in all these cases seems to support the view that the disease is a deficiency disease due to improper and unbalanced diet defective in proteins leading, in the later stages of the disease, to a deficiency of both mineral elements and anti-anæmic factor.

The authors are fully aware of the meagre details contained in this paper. Its chief purpose is to prove the existence of pellagra in southern India and of the efficacy of a protein-rich diet combined with liver in the cure of the disease.

Our thanks are due to Dr. P. Kutumbiah, for case 5; to Dr. P. Kesavaswamy, L.M.S., radiologist, for the photographs; to Dr. V. K. Narayana Menon and Dr. D. Narayana Rao for the biochemical investigations, and to Dr. P. Ramachandra Rao, for the pathological report of the specimen of the skin.

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[* In four out of the five cases in which the hæmoglobin percentage is given, it is a multiple of ten. This makes one wonder whether a very accurate method of hæmoglobin estimation was used. In these circumstances, or in any circumstances, we consider it rather bold to conclude that in one case the 'anti-anæmic factor' was absent (assuming that they mean 'absent' and not 'present' as the sentence reads), solely on the grounds that a marked degree of hyperchromia was demonstrated, and to conclude that mineral deficiency is a factor in the ætiology of pellagra on the grounds of finding a slight degree of hypochromia in two cases.—Editor, *I. M. G.*]

A NOTE ON CASES OF TYPHUS FEVER IN BURMA AND THEIR DISTRIBUTION

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Covell (1936) in an article in the *Indian Journal of Medical Research* gives an excellent review of the literature on typhus fever in India and mentions that the disease was known to be endemic in the trans-Indus districts of Yusufzai and Hazara and in the Himalayan hill tracts, more especially in Kulu Valley, as far back as 1894. Sir Leonard Rogers (1908) in his well-known book *Fevers in the Tropics* gives a small chapter on typhus fever. In this he states 'the annual death rate of 200 and upwards per thousand which used to occur in the insanitary and overcrowded jails of the Punjab even as late as 1878 was attributed and apparently with good reason, in part, to the prevalence of typhus fever by some experienced medical officers'. It is therefore clear that cases of typhus fever in India were suspected on clinical grounds in the neighbourhood of 60 years ago. In comparatively recent years a good deal of interest has been focused on the prevalence of this disease in India since Megaw and his collaborators (1917—28) contributed a series of articles in the *Indian Medical Gazette.*

As a result of this study it has been established that the disease is widely prevalent in India and not particularly confined to certain tracts or localities. The fact that it was not diagnosed for so long was due to lack of precise laboratory tests and inconstancy of clinical manifestations. In recent years gradual development of diagnostic procedures and employment of *Bacillus proteus,* 'X19' and its variants for the Weil-Felix test have marked a step forward in correct diagnosis of typhus fevers.

In Burma when the first case was diagnosed in 1932 by Kundu (1932) at the Rangoon General Hospital on clinical grounds and subsequently confirmed by agglutination test, it was considered that the infection probably was imported from Malaya where it had been recognized earlier and epidemics had occurred in the past. The second case, reported by Martin and Anderson (1933) a year after, contracted the disease at Thayetmyo in Upper Burma while touring in the jungles. A history of insect bite was available and the patient's serum reacted against the 'K' type of proteus 'X19'. Serologically this case was different from the first and came under the group 'scrub typhus' described by Fletcher (1931). The district of Thayetmyo in Upper