A THESIS
BY
A DESCRIPTION OF
ONE HUNDRED AND FOURTEEN
CASES OF FEVER
OF THE
TYPHUS GROUP SEEN IN
CALCUTTA
WITH A NOTE ON THE
PATHOLOGICAL FINDINGS,
A SURVEY OF
THE INDIAN LITERATURE
AND A
SHORT SURVEY
OF THE
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A DESCRIPTION OF ONE HUNDRED

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LITERATURE AND A SHORT

SURVEY OF THE GENERAL

LITERATURE.
HISTORICAL INTRODUCTION.

As the medical historian Hirsch (122) has said, "The history of typhus is written in those dark pages of the world's story which tell of the grievous visitations of mankind by war, famine and misery".

Murchison (208) discusses the genesis of the name typhus; τὸφος means smoke or stupeur, and it was used by Hippocrates to define a confused state of the intellect with a tendency to stupeur. Murchison considered that some descriptions of fever in Hippocrates' book on epidemics closely resemble typhus. He also considers that it is possible that typhus was one of the diseases referred to in sacred writings. During the first fifteen centuries of the Christian era, numerous epidemics of contagious fever occurred under circumstances of over crowding and famine. But Murchison says the descriptions of the Greek, Latin and Arabian physicians are not sufficiently precise to warrant us asserting that the fever in question was typhus. He considers however, that typhus was probably seen in Athens during the siege.

Hirsch (122) summarizes the history of typhus fever in Europe. He considers that the earliest reference to an outbreak which resembles
Typhus is an eleventh century epidemic in the monastry of La Cava near Salerno in 1303. He refers to Fracastorius (98) of Verona's "De Contagione et Contagiosis Morbis" in 1584 as the earliest description reliably recognised as typhus. Typhus was said to be present amongst the troops at the siege of Granada in 1489. In Spain it was named Tabardillo from the fancied resemblance of the florid rash to the coarse cape (tabardo) such as the country people wore, particularly to such a cape of a scarlet colour. Hirsch's summary of typhus in Europe recalls most of the less well-known as well as the prominent campaigns in European history during the past five centuries. Typhus appeared in England during the civil war. It was one of the prominent causes of death in the thirty years war between 1619 and 1648. It was seen not only on the field of battle or in the camp, but also in the courts of law when the jails disgorged some of their lice-infested unfortunates, as at the Black Assizes at Cambridge in 1552, and at the Old Bailey in London in 1750. MacArthur (166) gives a graphic picture of typhus in Britain in days of old.

Napoleon's "Grande Armee" retreating from Russia was attacked by typhus as well as by "General Winter".

KER (136) RECALLS THAT, "IN EDINBURGH DURING THE YEARS 1862-1871 NO LESS THAN 2824 CASES OF TYPHUS FEVER WERE TREATED IN THE ROYAL INFIRMARY ALONE" ....... "IN THE OLD DAYS THE STUDENTS AND RESIDENT PHYSICIANS OF THE EDINBURGH ROYAL INFIRMARY HAD A MOST PRACTICAL ACQUAINTANCE WITH THE FEVER, A LARGE PROPORTION OF THEM CONTRACTED IT, MANY DIED OF IT".

SIR HENRY LITTLEJOHN (161) IN 1898 REFERS TO THE DECREASE IN THE INCIDENCE OF TYPHUS IN EDINBURGH FOLLOWING THE COMPULSORY NOTIFICATION OF THE DISEASE.

AS MURCHISON (208) HAS SAID, "A COMPLETE HISTORY OF TYPHUS WOULD BE THE HISTORY OF EUROPE DURING THE LAST THREE AND A HALF CENTURIES".

IN THE FIRST GREAT WAR THERE WAS A SEVERE OUTBREAK IN SERBIA IN 1914-15. IT WAS PRESENT ON THE RUSSIAN FRONT ALSO IN POLAND. DURING THE SECOND GREAT WAR TYPHUS EXACTED ITS TOLL, BUT ITS POWER WAS ON THE WANE, THE WEAPONS TO CONTROL EUROPEAN TYPHUS HAD BEEN FORGED. ALLEN AND SPITZ (2) MENTION THAT IN THE SECOND WORLD WAR, NOT A SINGLE AMERICAN SOLDIER DIED OF LOUSEBOURNE TYPHUS.
BEFORE TYPHOID FEVER AND RELAPSING FEVER WERE RECOGNISED AS SEPARATE ENTITIES, THESE TWO DISEASES WERE INCLUDED IN THE FEVERS DIAGNOSED AS TYPHUS. GERHARD (103) IN 1837 AND STILLE (302) IN 1838 WERE THE FIRST TO DISTINGUISH THE TYPHUS-LIKE TYPHOID FEVER BY CLINICAL OBSERVATION. IN 1843 HENDERSON (120) DISTINGUISHED RELAPSING FEVER AS A SEPARATE ENTITY. WHAT WAS LEFT WAS A FEVER ASSOCIATED WITH POVERTY, STARVATION, FILTH AND OVERCROWDING.

ACCORDING TO ZINNSER AND BAYNE JONES (356), UNTIL 1917 NO DISEASE WAS RECOGNISED WHICH RESEMBLED OR WAS RELATED TO TYPHUS FEVER. MURCHISON (208) IN 1862 HAD EMPHASISED ITS CONTAGIOUSNESS. IN 1909 NICOLLE, COMTE AND CONSEIL (223) PROVED THAT TYPHUS COULD BE TRANSMITTED BY LICE, AND THIS WAS CONFIRMED IN 1910 BY RICKETTS AND WILDER (264).

IN 1898 BRILL (34) IN NEW YORK, DESCRIBED THE DISEASE WHICH BEARS HIS NAME. HE WAS LATER (35) ABLE TO SHOW THAT IT WAS NOT UNCOMMON AND BY 1910 HAD COLLECTED 221 CASES. ANDERSON AND GOLDBERGER (3) WERE ABLE TO SHOW BY CROSS-IMMUNITY TESTS WITH MONKEYS THAT BRILL'S DISEASE WAS A FORM OF TYPHUS.

THE DISCOVERY OF THE CASUAL RICKETTSIA AND OF THE WEIL-FELIX REACTION.

RICKETTS AND WILDER (264) IN 1910, AND WILDER
In 1911, observed certain minute bodies in the cells lining the gut of lice which had fed on typhus patients. These they considered were the organisms causing typhus fever. Von Prowazek (322) working in the Serbian epidemic of 1915 described similar micro-organisms. These findings were confirmed by other workers and the generic name of "Rickettsia" was applied to them by Da Rocha-Lima (266) in 1916. The particular organism proved to be responsible for the epidemic typhus in Europe was given the specific name "Prowazeki". These names honoured Ricketts and Von Prowazek who both died of typhus during the pioneer investigations.

In 1909 Wilson (337) published his observations made in Belfast, on a certain organism which he isolated from the faeces of a patient with typhus fever and which he named Bacillus "U". These important observations were to revolutionize the diagnosis of many fevers not then considered to be related to typhus. The reaction was developed by Weil and Felix (329) & (330) who in 1916, during the First World War, isolated several organisms of the Proteus group from patients with typhus fever in Poland. One of these, with a laboratory label "X.2", was used to confirm serologically the diagnosis of typhus. Another strain "X.19" discovered later,
GAVE THE MOST SATISFACTORY RESULTS, AS IT WAS AGGLUTINATED BY HIGHER DILUTIONS OF TYPHUS SERUM, AND IN SOME CASES WAS AGGLUTINATED WHEN "X.2" WAS NOT.

THE XK STRAIN, AS FELIX AND RHODES (91) REPORT, ORIGINATED AS FAR AS COULD BE ASCERTAINED WHEN AN X.19 STRAIN WAS SUPPLIED TO THE BLAND-SUTTON INSTITUTE IN 1921 BY THE NATIONAL COLLECTION OF TYPE CULTURES AS A TYPICAL STRAIN OF BACILLUS PROTEUS X.19; AND AS SUCH WAS BROUGHT OUT TO THE STRAITS SETTLEMENTS BY DR. A. N. KINGSBURY IN 1923. IT WAS FOUND TO HAVE ALTERED BIOLOGICAL REACTIONS AND IT WAS FOUND THAT IT WAS AGGLUTINATED BY SERA FROM CASES OF FEVER WHICH DID NOT AGGLUTINATE THE ORIGINAL X.19 OR X.2.

ANIMAL EXPERIMENTS.

RICKETTS (263) IN 1906 WAS THE FIRST TO DEMONSTRATE THE EXPERIMENTAL TRANSMISSION OF RICKETTSIAL DISEASE BY INOCULATION OF GUINEA PIGS AND MONKEYS WITH THE BLOOD OF CASES OF ROCKY MOUNTAIN FEVER. AT THE SAME TIME AS WILSON'S DISCOVERY, NICOLLE (220) SUCCESSFULLY INOCULATED AN ANTHROPOID APE, AND ANDERSON AND GOLDBERGER (3) INOCULATED LOWER MONKEYS WITH THE DISEASE. VERY SHORTLY AFTER NICOLLE, CONSEIL AND CONOR (224) SUCCESSFULLY INFECTED GUINEA PIGS WITH
HUMAN BLOOD, AND VIRUS FROM LICE. NEIL (216) IN 1917 REPORTED THE SCROTAL SWELLINGS IN MALE GUINEA PIGS INOCULATED WITH MEXICAN TYPHUS. IN 1919 NICOLLE AND LE BAILLY (228) IN EUROPE FOUND THAT GUINEA PIGS COULD BE INFECTED WITHOUT SCROTAL REACTIONS, AND IN SOME CASES COULD BE INFECTED WITHOUT ANY OBVIOUS SIGNS OF DISEASE DEVELOPING, THE SO CALLED "INAPARENTÉ" INFECTION. THEY ALSO FOUND THAT RATS DEVELOPED AN "INAPARENTÉ" INFECTION ONLY. GUINEA PIGS AND RATS ARE BOTH EXTREMELY FERTILE AND THEIR CONVENIENT SIZE MAKES THEM SUITABLE ANIMALS FOR EXPERIMENTAL PURPOSES. MONKEYS ARE OCCASIONALLY USED; AS BY LEWTHWAITE AND SAVOOR (149) FOR EXAMPLE, TO DEMONSTRATE SKIN LESIONS.

GUINEA PIGS, ACCORDING TO ZINNSER AND BAYNE JONES (356) ARE THE CHIEF INSTRUMENTS FOR TYPHUS EXPERIMENTATION AND DIFFERENTIATION. NAGAYO (212) DISCOVERED THAT THE INTRODUCTION OF INFECTED MATERIAL INTO THE ANTERIOR CHAMBER OF THE RABBIT'S EYE RESULTED IN AN IRIDO-CYCLITIS. RICKETTSIAE WERE FOUND IN DESCEMENTS MEMBRANE. THIS PROCEDURE WAS FOUND OF VALUE FOR DEMONSTRATING AND IDENTIFYING THE CASUAL RICKETTSIAE OF TSUTSUGAMUSHI FEVER. OTHER ANIMALS MAY BE USED FOR LABORATORY PROCEDURES, MICE FOR EXAMPLE, AND DURING THE SECOND WORLD WAR,

ZARAFONETIS (352) FOUND THAT THE EGYPTIAN RODENTS,
GERBILLUS PYRAMIDUM and GERBILLUS GERBILLUS, WERE A SUITABLE SUBSTITUTE FOR WHITE MICE FOR WORK WITH SCRUB TYPHUS. BUCKLAND AND CO-WORKERS (39) USED COTTON RATS IN THE MASS PRODUCTION OF A SCRUB TYPHUS VACCINE.

A SUMMARY OF THE "TYPHUS-LIKE" FEVERS.


TSUTSUGAMUSHI DISEASE.

THIS DISEASE HAS BEEN WELL KNOWN IN JAPAN FOR A THOUSAND YEARS ACCORDING TO MANSON-BAHR (171). BLAKE, MAXCY, SADUSK, KOHLS AND
BELL (24) SUMMARIZE THE GROWTH OF OUR KNOWLEDGE OF TSUTSUGAMUSHI UP TO 1946.

IT WAS NOT UNTIL 1924 THAT NAGAYO (210) AND HIS COLLEAGUES SAW THE RICKETTSIAE CAUSING THE DISEASE, AND NOT UNTIL 1930 WERE THEY ABLE TO DEMONSTRATE IT IN SUFFICIENT NUMBERS TO DEFINE IT, AND NAME IT "RICKETTSIA ORIENTALIS". THE PRIOR CLAIM OF HAYASHI (114) IS NOT NOW ACCEPTED, AND AS SELLARDS (281) WAS ABLE TO GROW THE MICRO-ORGANISM WHICH HE ISOLATED, ON ORDINARY BACTERIOLOGICAL MEDIA, MOST WORKERS CONSIDER THAT IT CANNOT HAVE BEEN A RICKETTSIA. OGATA (231) IN 1931 WAS ABLE TO DEMONSTRATE A RICKETTSIA AFTER PROPAGATION IN RABBITS TESTES. THIS HE CONSIDERED TO BE THE PROBABLE CAUSATIVE ORGANISM OF TSUTSUGAMUSHI. BLAKE (24) AND HIS COLLABORATORS HAVE POINTED OUT, THAT SOME OF THE EXPERIMENTAL RESULTS ON MONKEYS, PRIOR TO THE PRECISE IDENTIFICATION OF THE CAUSAL AGENT IN 1930, ARE IN SOME MEASURE DOUBTFUL BECAUSE OF LACK OF CONTROL RESULTS AND STERILITY TESTS ON INOCULATION MATERIAL. IT SEEMS FAIRLY PROBABLE HOWEVER, THAT KITASHIMA AND MIYAJIMA (137) IN 1918 WERE ABLE TO TRANSMIT THE DISEASE TO MONKEYS. AS LONG AGO AS 1899 TANAKA (307) HAD BEEN CONVINCED OF THE AETIOLOGICAL RELATIONSHIP OF A MITETE TO TSUTSUGAMUSHI DISEASE. THE SEASONAL DISTRIBUTION OF THE CASES OF THE DISEASE
CORRESPONDED WITH THE APPEARANCE AND DISAPPEARANCE OF THE MITES, AND THE INFLUX OF PEASANTS INTO THE ENDEMIC AREAS. THE EARLIER INVESTIGATIONS ON THE MITE IN JAPAN ARE ALSO CONFUSING BECAUSE OF THE LACK OF ACCURATE CLASSIFICATION OF THE INSECTS. THE TROMBICULA AKAMUSHI WAS STUDIED BY MIYAJIMA AND OKUMURA (203) IN 1917, AND BY NAGAYO (211) AND HIS COLLABORATORS IN 1921, AND BY OTHER WORKERS.

BLAKE AND HIS COLLABORATORS (24) DESCRIBE FULLY THE LIFE CYCLE OF THE MITE TROMBICULA AKAMUSHI. IT IS A MINUTE YELLOWISH RED INSECT, 0.32-0.43 m.m. IN LENGTH, IT DEVELOPS IN THE SOIL, AND LAYS EGGS LATE IN MAY FROM WHICH LARVAE EMERGE TO FEED ON VOLES, OR OCCASIONALLY MAN. HAVING FED THEY DROP OFF AND RETURN TO THE SOIL WHERE THEIR FURTHER DEVELOPMENT TAKES PLACE.

OFFERED IN 1930 BY KAWAMURA AND IMAGAWA (130) WHO RECOVERED THE VIRUS FROM NATURALLY INFECTED FIELD MICE (VOLES).

**TSUTSUGAMUSHI DISEASE IN THE JAPANESE EMPIRE.**

HATORI (112) CONCLUDED THAT A SIMILAR DISEASE WAS PRESENT IN FORMOSA, THOUGH LESS SEVERE, AND WITH AN INCIDENCE FROM APRIL TO NOVEMBER. IT ALSO APPEARED AWAY FROM THE RIVER VALLEYS. THE TROMBICULA AKAMUSHI WAS FOUND ON MANY SPECIES OF RODENTS. KAWAMURA AND YAMAGUCHI (131) CONFIRMED AND EXTENDED THESE OBSERVATIONS. IN SOME OF THE PESCADORES ISLANDS THIRTY MILES WEST OF FORMOSA, KAWAMURA AND YAMAMIYA (132) REPORTED IN 1939, THAT TSUTSUGAMUSHI DISEASE, IN MANY RESPECTS SIMILAR TO THE DISEASE IN FORMOSA, WAS ENDEMIC.

**TSUTSUGAMUSHI DISEASE OUTSIDE JAPAN.**

IN 1909 SCHUFFUER (279) AND IN 1910 SCHUFFUER AND WASHSMUTH (280) DREW ATTENTION TO THE PSEUDO-TYPHOID OF DELI, IN SUMATRA. A PRIMARY LESION WAS ALWAYS SEEN IN EUROPEANS. WALCH AND KEUKENSCHRIJVER (323) WERE ABLE TO ASSOCIATE INFECTION WITH THE DISTRIBUTION OF TROMBICULA DELIENSIS. THE NAME "MITE" FEVER (MITJEKOORTS) WAS SUGGESTED IN 1926. KOUWEENAR AND WOOLF (141) CONSIDERED THAT SUMATRAN MITE
FEVER WAS MORE CLOSELY RELATED TO THE SCRUB VARIETY OF TROPICAL TYPHUS THAN TO TSUTSUGAMUSHI FEVER. THESE DISTINCTIONS WERE DEPENDENT ON REACTIONS IN GUINEA PIGS AFTER INOCULATION AND THE SUPPOSED CLINICAL DIFFERENCES BETWEEN TSUTSUGAMUSHI DISEASE AND SCRUB TYPHUS. THESE DIFFERENCES ARE NOT NOW CONSIDERED TO BE REAL.

IN MALAYA THE FIRST CASE WAS REPORTED BY DOWDEN (71) IN 1915. FLETCHER AND LESSLAR (95) BEGAN REPORTING THEIR WORK ON THE TYPHUS-LIKE FEVERS OF MALAYA IN 1925, AND THEIR WORK WAS AMPLIFIED BY LEWTHWAITE (146) IN 1930. ONE VARIETY OF THE DISEASE LATER CONSIDERED TO BE TSUTSUGAMUSHI FEVER WAS ASSOCIATED WITH THE PALM TREES AND THE TALL COARSE GRASS (LA LANG) IN WHICH A PROLIFIC WILD RAT POPULATION LIVED. IN THE EARS OF THESE RATS, LARGE NUMBERS OF LARVAE TROMBICULA DELIENSIS WERE FOUND. A FEW TROMBICULA AKAMUSHI WERE FOUND BY GATER (101). THIS WAS CONFIRMED BY EWING (79). LEWTHWAITE AND SAVOOR (151) ISOLATED IN GUINEA PIGS, TWO STRAINS OF RICKETTSSIAE FROM WILD RATS, TRAPPED IN AN ENDEMIC AREA. AFTER FURTHER WORK THEY REGARDED THE WILD BROWN RAT (SPECIES NOT GIVEN) AS THE RESERVOIR OF THE CAUSATIVE AGENT OF TSUTSUGAMUSHI DISEASE IN MALAYA AND THE LARVAE TROMBICULA DELIENSIS ALMOST CERTAINLY AS THE
VECTOR. EXPERIMENTAL PROOF OF THE EXISTENCE OF THE CAUSATIVE AGENT IN MITES OF ANY SPECIES IN MALAYA IS STILL LACKING (24). LEWTHWAITE AND SAVOOR (155) WERE ABLE TO PRODUCE EVIDENCE BY CROSS-IMMUNITY TESTS, THAT TSUTSUGAMUSHI AND RURAL TYPHUS WERE IDENTICAL.

IN AUSTRALIA, SMITHSON (289) IN 1910 HAD REPORTED FEVERS OF UNKNOWN ETIOLOGY FROM THE "MOSSMAN" DISTRICT. IN 1934 LANGAN AND MATTHEWS (144) FOUND THAT SOME SERA FROM SUCH CASES AGGLUTINATED OXK IN HIGH DILUTION. IN 1940-41 HEASLIP'S (115) INVESTIGATIONS LED HIM TO BELIEVE THAT CASES OF WHAT WAS CALLED ELSEWHERE, "MITE FEVER", "SCRUB TYPHUS", "K TYPHUS" OR "TSUTSUGAMUSHI" OCCURRED IN NORTH QUEENSLAND. HE OBTAINED A STRAIN FROM WHICH AN ASSISTANT CONTRACTED THE DISEASE, AND WAS SATISFIED THAT 15 OUT OF 125 RATS AND 1 OUT OF 52 BANDICOOTS WERE DEFINITELY NATURALLY INFECTED WITH TSUTSUGAMUSHI DISEASE. THE VECTOR IS STILL ONLY SUSPECTED TO BE TROMBICULA DELIENSI IN AUSTRALIA.

IN THE MANDATED TERRITORY OF NEW GUINEA, GUNThER (108) INVESTIGATED A DISEASE VERY SIMILAR TO TSUTSUGAMUSHI, SEROLOGICALLY OXK.

IN INDO-CHINA, CLINICAL REPORTS WERE PUBLISHED IN 1908 BY YERSIN AND VASSAL (351) AND LATER BY OTHER OBSERVERS. THE FIRST SEROLOGICALLY PROVED CASE WAS IN 1932 AND A STRAIN WITH
SIMILAR REACTIONS TO RICKETTSIA ORIENTALIS WAS ISOLATED BY SOUCHARD, MARNEFFE, LIEOU AND VIELLE (295). THE VECTOR AND ANIMAL RESERVOIR ARE UNKNOWN.

IN 1933 MARTIN AND ANDERSON (175) REPORTED A CASE PROBABLY SCRUB TYPHUS FROM BURMA. BOYD (28) WAS ABLE TO REPORT 35 CASES OF SCRUB TYPHUS DIAGNOSED ON SEROLOGICAL GROUNDS IN 1934 FROM ARMY CASES NOTIFIED IN INDIA.

PANDALAI (235) FROM SOUTH INDIA REPORTED TWO CASES, POSSIBLY SCRUB TYPHUS, IN 1936. KAPILA AND MAITRA (129) REPORTED ONE CASE FROM BHAMO ON THE BURMA-CHINESE BORDER, AN AREA WHERE SCRUB TYPHUS WAS LATER TO BE ENCOUNTERED BY THE AMERICANS AND CHINESE DURING THE SECOND WORLD WAR, AND WHERE MACKIE (169) AND HIS COLLABORATORS WERE TO CARRY OUT THEIR INVESTIGATIONS. MANY REPORTS WERE MADE OF OUTBREAKS OF SCRUB TYPHUS DURING THE SECOND WORLD WAR, FROM ASSAM, BURMA, BENGAL, CEYLON AND NEW GUINEA.

MURINE TYPHUS.

A LESS VIRULENT FORM OF TYPHUS FEVER FOUND ALL OVER THE WORLD IS CONVEYED FROM ITS ANIMAL RESERVOIR THE RAT, TO MAN BY THE RAT FLEA. THE RATS BELONG TO THE SUB-FAMILY 'MURINAE', HENCE THE NAME "MURINE TYPHUS". MURINE TYPHUS IS COMMON AROUND THE MEDITERRANEAN COAST. THE
TYPHUS "ENDEMIQUE BENIN" OF PLAZY, MARCON AND CARBONI (252) AND THE "FIEVERE NAUTIQUE" OF MARCHANDER AND BIDEAU (176) SEEN IN TOULON, HAVE BEEN INVESTIGATED BY VARIOUS WORKERS WITH THE ISOLATION OF THE SPECIFIC MURINE RICKETTSIAE. MURINE TYPHUS WAS ENCOUNTERED IN THE MIDDLE EAST DURING THE SECOND WORLD WAR BY VAN ROOYEN AND BEARCROFT (319). A MURINE STRAIN HAS BEEN ISOLATED FROM RAT FLEAS IN ATHENS BY LEPINE, CAMINOPTEROS AND PANGALOS (157), AND FROM RATS AND RAT FLEAS IN ALEXANDRIA BY PANAYOLALAN (234).

REITLER, BTESH AND MARBERG (261) CONSIDER ENDEMIC (OR MURINE TYPHUS) TO BE COMMON IN PALESTINE.

MAISTER AND MILLER (172) REPORT A CASE AND GEAR AND DE MEILLON (102) ISOLATED A MURINE STRAIN IN NATAL IN 1939.

PIJPER AND DAU (247) REPORT THAT RAT FLEA TYPHUS OCCURS IN VARIOUS PARTS OF THE UNION OF SOUTH AFRICA.

DURING THEIR INVESTIGATIONS ON TROPICAL TYPHUS IN MALAYA, LEWTHWAITE AND SAVOOR (154) STUDIED IN LABORATORY ANIMALS ONE FORM OF TROPICAL TYPHUS, THE URBAN OR SHOP TYPHUS AS IT WAS CALLED THERE. THE SCROTAL REACTIONS IN GUINEA PIGS AND OTHER RESPONSES WERE CONSISTENT WITH MURINE TYPHUS. LATER THEY (154) SHOWED THAT SEROLOGICALLY AND ON CROSS-IMMUNITY TESTS
IT WAS DISTINCT FROM TSUTSUGAMUSHI DISEASE.

THEY (155) LATER CONSIDERED IT TO BE THE FLEA BORNE ENDEMIC DISEASE FOUND THE WORLD OVER.

IT HAS BEEN REPORTED FROM INDO-CHINA BY DELBORE (67).

LIU AND CHUNG (162) FROM PEIPING, AND RAYNAL (259) FROM SHANGHAI REPORT THE ISOLATION OF MURINE VIRUSES ON THE CHINA COAST. MAXCY (177) & (178) AND DYER AND CEDER, RUMREICH AND BADGER (73) HAVE SHOWN THAT THE MILD ENDEMIC TYPHUS OF THE SOUTHERN UNITED STATES IS SPREAD BY THE RAT FLEA.

FELIX (87) CONSIDERS THAT HONE'S (123 & 124) DISEASE FROM PORT ADELAIDE IN AUSTRALIA, AND LATER REPORTED FROM PERTH, MELBOURNE, SYDNEY AND BRISBANE, IS MURINE TYPHUS, AS IS THE "ENDEMIC TYPHUS OF GREECE, SYRIA, MANCHURIA, THE PHILLIPINES AND HAWAI".

IN BURMA MURINE TYPHUS HAS BEEN SUSPECTED ON CLINICAL AND SEROLOGICAL GROUNDS IN KUNDU'S (142) CASE FROM RANGOON, AND ONE OF SONI'S (294) CASES FROM UPPER BURMA. MAITRA AND SEN GUPTA'S (173) REPORT ON THE RESULT OF WEIL FELIX TESTS OF SERA FROM ALL OVER BURMA, SUGGESTS THAT IT POSSIBLY MAY BE PRESENT, IN THE COUNTRY DISTRICTS, AS WELL AS IN THE TOWNS.

IN BOMBAY, PATEL'S (243 & 244) SEROLOGICAL INVESTIGATIONS SUGGESTED THE POSSIBILITY OF
MURINE TYPHUS IN THAT PORT.

COVELL (54) in 1936 was able to report the isolation of a virus from the brain of a wild rat in Simla. COVELL and MEHTA (55) were able to transfer it to another rat by a rat flea (Xenophylla Cheopis). Four years later WEBSTER (326) reported that no strains had been isolated from any human case.

BOYD (28) considered that the cases seen in Bangalore, serologically 0x19, were probably endemic flea typhus. NICHOLIS (219) in his remarks at an Army Conference on typhus in Ceylon in 1944, mentioned that Dr. E. M. WIJERAMA (334) described a case of urban typhus (X19) in 1938. The City Microbiologist DR. E. K. WOLFF (342), injected guinea pigs with an emulsion of rat fleas (X. Astia, X Cheopis) caught in Colombo. This produced fever and scrotal changes typical of the "X19" type infection, and smears taken from the tunica vaginalis showed rickettsiae.

GOYAL (105) was able to demonstrate an epizootic of a rickettsial infection in Calcutta wild rats from June 1937 to December 1938 when it died out. Further investigations were negative. The position in India today is
THAT VERY LITTLE WORK HAS BEEN DONE TO CONFIRM
AN IMPRESSION THAT MURINE TYPHUS IS PRESENT IN
THE LARGE PORTS AND ELSEWHERE.

TABARDILLO IN MEXICO HAS BEEN SHOWN BY
MOOSER (204) TO BE MURINE TYPHUS.

BRILL'S DISEASE, WHICH IS A MILD INFEC-
TION, WAS ON THAT ACCOUNT AT ONE TIME CON-
SIDERED TO BE ENDEMIC FLEA-BORNE TYPHUS.
ZINNSER'S (354) FINAL OPINION WAS THAT THE
CASES OF TYPHUS (BRILLS DISEASE) SEEN IN
THE NORTH ATLANTIC STATES WERE LATE RE-
CRUDESENCES OF CHILDHOOD INFECTION WITH
EUROPEAN TYPHUS. THIS OPINION WAS CONFIRM-
ED BY PLOTZ'S (253) SEROLOGICAL INVESTIGATION
WITH THE COMPLIMENT FIXATION TEST.

PINKERTON (248) DISCUSSES THE POSSI-
BILITY OF A MURINE STRAIN IN MAN BECOMING,
IN THE PRESENCE OF OVERCROWDING AND ABUND-
ANCE OF LICE, AN EPIDEMIC EXANTHEMATIC
TYPHUS, WITH ALTERATION IN THE TYPE OF
RICKETTSIA. "NO ONE", SAYS PINKERTON (248)
"HAS CHANGED THE TYPE OF THE RICKETTSIA
EXPERIMENTALLY, EVEN AFTER YEARS OF PASSAGE".
BUT HE QUOTES ZINNSER (355) WHO CONSIDERS
THAT A MURINE RICKETTSIA MAY BE SPREAD BY THE
LOUSE. MOOSER AND DUMMER (205) WERE ABLE TO
SHOW EXPERIMENTALLY THAT THIS COULD TAKE
PLACE.
GURBUKSH SINGH, (109) WHOSE CASES OF SCRUB TYPHUS IN BURMA WERE LIMITED TO A LOUSE-INFESTED UNIT, CONSIDERED IT WAS POSSIBLE THAT A CHANGE OF VECTOR MIGHT CHANGE THE STRAIN OF THE RICKETTSIA; THIS HOWEVER WAS ONLY A SPECULATION. FOR THE PRESENT, IT IS CONSIDERED THAT EVEN IF THE LOUSE SHOULD PASSAGE A MURINE VIRUS, THAT VIRUS RETAINS ITS MURINE CHARACTER. THERE IS NO EVIDENCE THAT RICKETTSIA ORIENTALIS CAN BE PASSAGED BY THE LOUSE.

ROCKY MOUNTAIN SPOTTED FEVER.

THE FIRST CASES OF THIS DISEASE WERE REPORTED IN 1904. HISTORY HAS LITTLE TO SAY ABOUT THE CONDITIONS BEFORE THE PENETRATION OF THE WHITE MAN TO THE AFFECTED AREAS. THIS FEVER OF THE OPEN COUNTRY WAS NOT ASSOCIATED IN MEN'S MINDS WITH TYPHUS UNTIL THE RICKETTSIAL CAUSATION OF BOTH WAS ESTABLISHED.

WOOLBACH (345) IN 1919, IN THE INTRODUCTION TO HIS CLINICAL AND PATHOLOGICAL STUDIES OF ROCKY MOUNTAIN SPOTTED FEVER, SUMMARIZED THE GROWTH OF OUR KNOWLEDGE OF THE DISEASE AND ITS SPREAD BY A TICK. THE AREAS WHERE THE INFECTED TICKS WERE FOUND WERE OFTEN SHARPLY LOCALIZED. THE MORTALITY VARIED VERY CONSIDERABLY. THE SO-CALLED WESTERN SPOTTED FEVER FROM THE BITTER ROOT VALLEY HAS A MORTALITY OF 70%. OUTSIDE THE BITTER ROOT VALLEY, WOOLBACH'S (345) MORTALITY
FIGURES ARE 7-13%. HOWEVER TOPPING'S (313) FIGURES OVER A TEN YEAR PERIOD SUGGEST THAT THESE FIGURES MAY REQUIRE A CERTAIN AMOUNT OF QUALIFICATION.

CLINICALLY IN ROCKY MOUNTAIN SPOTTED FEVER THERE IS A PROFUSE PETECHIAL OR PURPURIC RASH, WHICH BEGINS ON THE ANKLES, WRISTS AND FOREHEAD, LATER BECOMING GENERALIZED. THERE IS A TENDENCY FOR AREAS OF SKIN AND ALSO THE EXTERNAL MALE GENITALS, TO BECOME GANGRENOUS. THIS TENDENCY TO GANGRENE IS ALSO SEEN IN LABORATORY ANIMALS.

IN THE EASTERN UNITED STATES A VERY MILD SPOTTED FEVER IS SEEN, SUCH AS THE VERY MILD MINETOSA VARIANT, FIRST DESCRIBED BY REIMAN, ULRICH AND FISHER (260).

ZINNSER AND BAYNE-JONES (356) SPECULATE WHETHER THE PERSISTENT PASSAGE OF THE BITTER ROOT WESTERN STRAIN THROUGH DOGS AND DOG TICKS MAY HAVE CAUSED THE MILD EASTERN VARIANT. THE EASTERN AND WESTERN VARIETIES CROSS-IMMUNIZE WITH EACH OTHER.

SPENCER AND PARKER (298) HAVE SHOWN THAT AN INFECTED TICK WHICH HAS RECENTLY FED, CONTAINS VIRULENT IMMUNITY PRODUCING VIRUS, WHEREAS AN UNFED FASTING INFECTED TICK CONTAINS NON-VIRULENT IMMUNITY PRODUCING VIRUS. PARKER (238) STATES THAT THE VIRUS OF ANY INDIVIDUAL
INFECTED WOOD TICK OF A LOCAL TICK POPULATION, MAY VARY FROM A NON-IMMUNIZING, NON-DEMONSTRA-
BLE PHASE, TO THAT OF THE PREVAILING MAXIMUM LOCAL VIRULENCE. IN FASTING, HIBERNATING WOOD
TICKS, INFECTED WITH ROCKY MOUNTAIN FEVER VIRUS, OR ONE JUST EMERGING FROM HIBERNATION, THE
VIRUS IS USUALLY COMPLETELY INACTIVE OR IN A PHASE WHICH CAUSES "INAPPARENT" INFECTIONS
WHEN INJECTED INTO LABORATORY ANIMALS. HOWEVER, IF SUCH A TICK IS INCUBATED OR ALLOWED
TO INGEST BLOOD, THE VIRUS IS "REACTIVATED" AND SOON REACHES ITS MAXIMUM POTENTIAL VIRU-
LENCE. WHILE SUCH EVIDENCE IS OF INTEREST, IT IS NOT SUGGESTED THAT THE INDIVIDUAL
RICKETTSIAE MAY ALTER THEIR VIRULENCE PERM-
ANENTLY.

THE SEVERE SAO PAULO FEVER, ALSO SPREAD
BY A TICK, HAS BEEN SHOWN BY PARKER AND DAVIS (237) TO CROSS-IMMUNIZE COMPLETELY WITH THE
SEVERE WESTERN SPOTTED FEVER.

THE FIEVRE BOUTONNEUSE IS FOUND IN ALL COUNTRIES ALONG THE SHORES OF THE MEDITERR-
ANEAN, AND IN THE BALKANS, INCLUDING RUMANIA. THOUGH A MILD DISEASE, THE IMMUNOLOGICAL
STUDIES OF BADGER (10), HASS AND PINKERTON (111), AND BLANC AND BALTAZARD (23), HAVE
ESTABLISHED ITS GENERAL RELATIONSHIP WITH THE SPOTTED FEVERS, WHICH IT RESEMBLES IN HAVING
A TICK VECTOR. IT ALSO SHARES WITH TSUTSU-
GAMUSHI DISEASE THE ESCHAR AND LOCAL ADENO-
PATHY. LEWTHWAITE AND SAVOOR (155) WERE ABLE 
TO SHOW A DERMAL LESION IN ONE OF THEIR CASES 
OF TSUTSU-GAMUSHI DISEASE IN MALAYA TO PROFESSOR. 
M.CIVCA, OF BUCHAREST, WHO WAS FAMILIAR WITH 
THE "TACHE NOIR" OF FIEVRE BOUTONNEUSE. HE 
CONSIDERED THE DERMAL LESION OF THE TWO DIS-
EASES INDISTINGUISHABLE.

THE FIEVRE EXANThEMATIQUE OF MARSEILLES 
AND THE FEBBRE ERRUTIVA OF ITALY ARE CONSIDERED 
BY FELIX (87) TO BE IN THE SAME GROUP AS ROCKY 
MOUNTAIN SPOTTED FEVER. BALFOUR (12) CONSIDER-
ED THE TICK-BORNE TYPHUS OF KENYA TO BE FIEVRE 
BOUTONNEUSE. PIJPER AND DAU (247) AGREE.

THE TICK-BITE FEVER OF SOUTH AFRICA IS, 
ACCORDING TO PIJPER AND DAU (247), UNLIKE 
FIEVRE BOUTONNEUSE. THEY, AND PARKER AND DAVIS 
(237) HAVE SHOWN THAT THERE IS NO CROSS-
IMMUNITY WITH ROCKY MOUNTAIN SPOTTED FEVER AND 
TICK-BITE FEVER.

PIJPER AND DAU (247) HAVE POINTED OUT 
THAT THE SOUTH AFRICAN LOUSE-BORNE TYPHUS AND 
FLEA-BORNE ENDEMIC TYPHUS DO NOT EXACTLY RES-
SEMBLE THEIR COUNTERPARTS ELSEWHERE.

IN INDIA, THOUGH MEGAW (187) ON CLINIC-
CAL AND EPIDEMIOLOGICAL GROUNDS SUGGESTED THE 
SIMILARITY OF CASES OF TYPHUS IN INDIA WITH 
ROCKY MOUNTAIN SPOTTED FEVER AND SUGGESTED THE
TICK AS A VECTOR, NO EVIDENCE WAS PRODUCED UNTIL HEILIG AND NAIDU (119) PUBLISHED RESULTS OF COMPLIMENT FIXATION TESTS, CARRIED OUT BY TOPPING, ON THE SERA OF FOUR OF THEIR CASES FROM MYSORE IN SOUTHERN INDIA. THESE SERA CONTAINED ANTIBODIES MORE NEARLY RELATED TO ROCKY MOUNTAIN SPOTTED FEVER THAN TO EITHER EPIDEMIC OR ENDEMIC (MURINE) TYPHUS.

TRENCH FEVER OR WOLHYNIA FEVER.

TRENCH FEVER IS A MILD BLOOD INFECTION COMMUNICATED FROM MAN TO MAN BY THE LOUSE. THE DISEASE HAS NOT REAPPEARED SINCE THE FIRST WORLD WAR. PINKERTON (248) CLASSES THE CAUSAL RICKETTSIA WOLHYNICA AS AN EXTRA-CELLULAR RICKETTSIA-LIKE PATHOGEN.

THE 'Q' FEVER.

THIS FEVER HAS NOW BEEN EXTENSIVELY STUDIED IN AUSTRALIA WHERE IT WAS FIRST REPORTED FROM BRISBANE BY DERRICK (68), AND IN AMERICA. ACCORDING TO FINDLAY (94), BOTH AUSTRALIAN AND AMERICAN WORKERS ARE NOW AGREED THAT 'Q' FEVER AS IT APPEARS IN THE TWO CONTINENTS IS IDENTICAL, AND THAT THE RICKETTSIAE RESPONSIBLE ARE STRAINS OF ONE AND THE SAME ORGANISM. PINKERTON (248) CONSIDERS THE CAUSAL RICKETTSIA BURNETI TO BE A FACULTATIVE, RATHER THAN AN OBLIGATE INTRACELLULAR PARASITE.
DURING THE SECOND WORLD WAR, 'Q' FEVER WAS FOUND TO BE PRESENT IN THE MEDITERRANEAN AREA AND IN THE BALKANS, AND HAS BEEN FULLY REPORTED BY ROBBINS, DINGLE (265) AND OTHER AMERICAN WORKERS. A SINGLE CASE OCCURRED IN PANAMA.

THE VECTOR IN TYPHUS AND THE TRANSMISSION OF THE INFECTION.

THE DISEASES CAUSED BY RICKETTSIAE ARE WIDESPREAD. THE KNOWN VECTORS ARE LICE, RAT FLEAS, TICKS OR LARVAL MITES.

IN MANY CASES IN INDIA THE VECTOR IS UNKNOWN. IN SOME CASES, BY ANALOGY, A PARTICULAR VECTOR IS SUSPECTED. PRECISE INFORMATION IS REQUIRED BEFORE CONCLUSIONS CAN BE DRAWN. ONE MIGHT EXPECT THAT RICKETTSIAE, WHICH ARE SO CLOSELY DEPENDENT ON LIVING TISSUES, WOULD BE CONSIDERABLY ALTERED IF THEY WERE INTRODUCED INTO A HOST BY MEANS OTHER THAN THE USUAL VECTOR. NEVERTHELESS, THE SPREAD OF THESE DISEASES BY DUST AND BY MEANS OTHER THAN BY INSECT VECTORS MUST BE CONSIDERED.

BLANC AND BALTAZARD (22) FOUND THAT THE VIRUS OF MURINE TYPHUS REMAINED INFECTIVE IN THE DRIED DEJECTA OF INFECTED FLEAS FOR 651 DAYS. THIS FACT HAD PREVIOUSLY BEEN NOTED IN THE CASE OF DRIED INFECTIVE LOUSE FAECES BY ARKWRIGHT AND BACOT (9) STARZYK (300) AND
FEJGIN (83). NICOLLE, GIROUĐ AND SPARROW
(226 & 227) HAVE SHOWN THAT OCCASIONALLY, THE
URINE OF EXPERIMENTALLY INFECTED RATS CONTAINS
SMALL AMOUNTS OF MURINE VIRUS. THEY WERE ABLE
TO PRODUCE IMMUNITY TO KNOWN MEXICAN STRAINS,
IN GUINEA PIGS INFECTED WITH THIS VIRUS FOUND
IN URINE. SPARROW (296) WAS ABLE TO PROVOKE
IMMUNITY TO MURINE VIRUS BY NASAL INSTILLATION
INTO RATS AND GUINEA PIGS OF EXTRACTS OF
BRAINS OF RATS INFECTED WITH THAT VIRUS. SHE
WAS ABLE TO REPRODUCE AN ATTACK OF TYPHUS IN
A YOUNG MAN BY INTRANASAL INSTILLATION OF THE
SAME VIRUS. SPARROW AND MARESCHALL (297)
WERE ABLE TO REPEAT THIS, USING THE SAME
MURINE STRAIN FROM AN INFECTED LOUSE. CON-
JUNCTIVAL INSTILLATION IN A HUMAN PATIENT
RESULTED IN A WEEK'S FEVER, THE WEIL-FELIX
BECOMING POSITIVE AND A SOLID IMMUNITY RES-
ULTING. SUBCUTANEOUS INJECTION OF THE VIRUS
IN ANIMALS PRODUCED A MORE SEVERE RESULT,
BUT THIS COULD BE REGULATED BY VARYING THE
DOSE. NICOLLE, GIROUĐ AND SPARROW (226 &
227) HAVE SHOWN THAT THE URINE OF RATS
EXPERIMENTALLY INFECTED WITH MURINE TYPHUS
MAY CONTAIN THE VIRUS. DYER, CEDER, WORKMAN,
RUMREICH AND BADGER (74) HAVE ALSO REPORTED
FROM BALTIMORE THE TRANSMISSION OF MURINE
TYPHUS TO RABBITS BY SMEARING INFECTED FLEAS
ON A SHAVED AREA OF THE ABDOMEN AND SCRATCHING
THROUGH THE SMEAR. ALSO IN A SIMILAR MANNER TO GUINEA PIGS BY SMEARING ON INFECTED FLEA FAECES AND SCRATCHING THROUGH THE SMEAR.

LABORATORY INFECTIONS ARE WELL KNOWN. MOOSER (204) REPORTED A MILD INFECTION AFTER INFECTIVE FLUID WAS SQUIRTED INTO THE FACE. VAN DEN ENDE AND HIS COLLABORATORS (317) REPORTED LABORATORY INFECTIONS AMONGST CO-WORKER ON SCRUB TYPHUS VACCINE PRODUCTION. 'Q' FEVER WAS FIRST REPORTED IN AMERICA AS A LABORATORY INFECTION. THE EXACT MECHANISM WAS NOT CLEAR, BUT FINDLAY (94) CONSIDERS INHALATION POSSIBLE.

VOILLE (320 & 321) REPORTS THE ORAL INFECTION WITH MURINE TYPHUS OF A PIG AND A DOG. STRONG (304) QUOTES NICOLLE (221) AND LE CHUITON (145) "WHO BELIEVE THAT MAN CAN BE INFECTED THROUGH THE INGESTION OF FOODS SOILED WITH RAT URINE, SINCE ANIMALS MAY BE INFECTED WITH MURINE TYPHUS THROUGH THE INGESTION OF MATERIALS CONTAINING THE VIRUS". STRONG IS OF THE OPINION THAT THERE IS GOOD EVIDENCE THAT THE VIRUS OF MURINE TYPHUS IS PRESENT IN THE URINE OF INFECTED RATS. THESE RESULTS ARE OF CONSIDERABLE INTEREST. THE INVESTIGATIONS HAVE BEEN MAINLY WITH MURINE VIRUS. EXPERIMENTAL RESULTS MUST OF COURSE BE APPLIED WITH CAUTION. NEVERTHELESS, I CONSIDER
That the transmission of typhus like fevers other than by an insect vector should be borne in mind. Particularly might "dust spread" be kept in mind by those investigating the disease in India and Burma where, during the dry weather, dust is ever present, and the daily bath of the majority of the inhabitants does not encourage personal ecto-parasites.

The relationship of Rickettsiae to typhus.

This need only be briefly mentioned. The opinion of Zinsser and Bayne-Jones (356) is that, "although there are still occasionally publications in which causation by true bacteria or by a filtrable virus is suggested, it is quite generally accepted by students of these diseases that they are caused by a class of micro-organisms spoken of as Rickettsia". The evidence for and against it is conveniently summarized by Topley and Wilson (312).

Anigstein (5) in his work on tropical typhus in Malaya, discusses the work of those who have suggested that the causal Rickettsia of typhus represents the parasitic stage of Proteus X in a complex life cycle. Anigstein (5) considered that his investigations supported this hypothesis, but his deductions are not
ENTIRELY CONVINCING.

PINKERTON (248) IS VERY CRITICAL OF EXPERIMENTS SUGGESTING THAT PROTEUS X IS THE CAUSE OF TYPHUS. THERE IS, AMONGST OTHER FACTS, NO CROSS-IMMUNITY TO INFECTIONS WITH PROTEUS X AND RICKETTSIAE PROWAZEKI. IT IS OF COURSE OBVIOUS THAT THE MERE DEMONSTRATION OF RICKETTSIAE IS NOT SUFFICIENT FOR THE DIAGNOSIS OF TYPHUS. AS FINDLAY (93) POINTS OUT, "UNEQUIVOCAL EVIDENCE THAT THE DISEASE IS TYPHUS CAN BE OBTAINED ONLY BY ISOLATION AND PASSAGE OF THE RICKETTSIAE IN SUSCEPTIBLE ANIMALS".

THE DEFINITION OF PATHOGENIC RICKETTSIAE IN THE LITERATURE.

PINKERTON (248) STATES THAT PRESENT OPINION CONSIDERS THAT THE RICKETTSIAE OCCUPY A POSITION INTERMEDIATE BETWEEN CERTAIN CYTOTOXIC BACTERIA AND THE VIRUSES. HE DEFINES THEM, (USING AS A BASIS CRITERIA SUGGESTED BY COWDRY (58 & 59) AND WOOLBACH (346)) AS "SMALL, OFTEN PLEOMORPHIC, GRAM-NEGATIVE, BACTERIUM-LIKE ORGANISMS, LIVING AND MULTIPLYING IN ARTHROPOD TISSUES, BEHAVING AS OBLIGATE INTRACELLULAR PARASITES AND STAINING LIGHTLY WITH ANALINE DYES WITH FEW EXCEPTIONS, CRITERIA ADEQUATE FOR CLASSIFICATION ON THE BASIS OF BIOLOGICAL PROPERTIES ARE AVAILABLE ONLY FOR
THOSE MEMBERS OF THE GROUP WHICH ARE PATHOGENIC FOR MAMMALS". HE CONSIDERS THESE IN MAN TO NUMBER THREE.

(1) RICKETTSIA PROWAZEKI, THE CAUSAL RICKETTSIA OF EXANTHEMATIC TYPHUS, WITH WHICH IS INCLUDED R. PROWAZEKI VAR. MOOSERI, THE CAUSAL RICKETTSIA OF MURINE TYPHUS.

(2) DERMACENTROXUS RICKETTSII, THE CAUSAL RICKETTSIA OF ROCKY MOUNTAIN SPOTTED FEVER (OR R. RICKETTSII).

(3) R. ORIENTALIS, THE CAUSAL RICKETTSIA OF TSUTSUGAMUSHI DISEASE.

PINKERTON (248) REGARDS R. BURNETI, THE 'Q' FEVER RICKETTSIA, AS A FACULTATIVE RATHER THAN AN OBLIGATE PARASITE; AND R. WOLHYNICA, THE TRENCH FEVER RICKETTSIA, AS AN EXTRA-CELLULAR RICKETTSIA-LIKE PATHOGEN. THE CHARACTERISTIC PROPERTIES OF THE PATHOGENIC RICKETTSIAE ARE MANIFEST WHEN THEY ARE INTRODUCED INTO GUINEA PIGS, OR SUITABLE LABORATORY ANIMALS. THEY PRODUCE CONSTANTLY A CHARACTERISTIC INFECTION, AND ARE DEMONSTRABLE INSIDE THE CYTOPLASM, OR EVEN INSIDE THE NUCLEI OF PARTICULAR CELLS.

IMMUNOLOGICAL TESTS ON GUINEA PIGS RECOVERING FROM RICKETTSIAL INFECTIONS, AS PINKERTON (248) SAYS, SERVE TO IDENTIFY THE PATHOGENIC RICKETTSIAE WITH GREAT PRECISION.
THE REASONS FOR THE LACK OF INFORMATION ON TYPHUS IN INDIA IN STANDARD TEXT BOOKS.

Owing to the lack of factual information from India, the standard text books of medicine have scanty reference to typhus and typhus-like fevers in that country and Burma.

Megaw (186, 187 & 189) was the first to attract attention to the fact that a typhus-like disease might be found in northern India. He stressed the differences between his disease and exanthematic typhus, and the similarities with the spotted fever of the Rocky Mountains. His early clinical work has had little support from Indian scientific investigators. The reasons are mainly financial. Laboratories and workers capable of carrying out the necessary research are few and far between. Specimens deteriorate rapidly and laboratory investigations in many cases just cannot be carried out in general practice. The poverty of the masses demands that medical investigations of the sick shall be of the simplest. The Indian of all classes is, like many other men, not anxious to go to hospital. In any case typhus-like fevers do not commonly present themselves in the clinics in large cities with teaching hospitals and laboratories. In addition, it must be admitted that the profession as a whole was
INCLINED TO BELIEVE THAT TYPHUS OR TYPHUS LIKE FEVERS WERE RARE IN INDIA.

AGGLUTINATION TESTS WHICH MIGHT HAVE GIVEN A CLUE TO THE PRESENCE OF TYPHUS ARE OFTEN NOT OF MUCH VALUE DURING THE HEIGHT OF THE ILLNESS, AND UNLESS SERIAL TESTS ARE CARRIED OUT MAY EVEN BE MISLEADING. EXPENSE USUALLY PRECLUDES MANY EXAMINATIONS. SUSPENSIONS HAVE OFTEN BEEN UNSTANDARDIZED AND UNRELIABLE. FEVER HAS SO OFTEN BEEN CONVENIENTLY EXPLAINED AS MALARIA, AND OFTEN THERE IS CO-INCEDENTAL MALARIAL PARASITAEMIA OR A RISE IN TYPHOID AGGLUTININS OF AN AMNESTIC NATURE RESULTING IN A FALSE DIAGNOSIS. EVEN IN THE LARGE CENTRES WITH TEACHING HOSPITALS ALL THE ABOVE DIFFICULTIES ARE PRESENT TO A GREATER OR LESSER DEGREE. IN ADDITION, THE OFTEN ILLUMINATING AUTOPSY IS USUALLY DENIED TO THE SEARCHER AFTER TRUTH.

NAPIER (213) GIVES A MORE UP-TO-DATE SUMMARY OF THE POSITION IN INDIA THAN THE OLDER TEXT BOOKS.

THE BRITISH ENCYCLOPEDIA OF MEDICAL PRACTICE (36) MENTIONS THE PROBABLE PRESENCE OF THE MITE TYPHUS IN BURMA. THE SECOND WORLD WAR PRODUCED MANY NOTABLE ADVANCES OF KNOWLEDGE IN MANY SPHERES. MUCH FRUITFUL KNOWLEDGE WAS GAINED BY MEDICINE IN INDIA, NOT THE LEAST BEING THE INFORMATION GAINED OF THE INCIDENCE
OF TYPHUS. MACKIE, (169) WITH HIS AMERICAN AND BRITISH COLLABORATORS, BY THEIR WORK ON TSUTSUGAMUSHI DISEASE IN ASSAM AND BURMA, DID MORE IN A YEAR THAN HAD BEEN DONE IN GENERATIONS.

THE WEIL FELIX REACTION.

THE TITLE OF WILSON'S (337) ORIGINAL PAPER WAS "HETERLOGOUS AGGLUTININS, MORE PARTICULARLY THOSE PRESENT IN THE BLOOD SERUM OF CEREBRO-SPINAL FEVER AND TYPHUS FEVER CASES". ELEVEN YEARS LATER, DISCUSSING THE REACTION, WILSON (338) SUGGESTED THAT TYPHUS RENDERED THE INTESTINE MORE PERMEABLE TO INTESTINAL MICRO-ORGANISMS, AND THAT CERTAIN STRAINS OF B. COLI OR ALLIED ORGANISMS INFECTED THE PATIENT, RESULTING IN THE PRODUCTION OF SPECIFIC AGGLUTININS FOR THE INFECTING ORGANISM, AND "PARAGGLUTININS" FOR OTHER ORGANISMS SUCH AS PROTEUS X.19 AND X.2, B. COLI, B. TYPHOSUS ETC. IN A FURTHER PUBLICATION IN 1927 HOWEVER, WILSON (339) CONSIDERED THAT THE WEIL FELIX REACTION WAS NOT PARAGGLUTINATION, AND DID NOT ACCEPT THE SUGGESTIONS MADE BY VARIOUS WORKERS THAT THE CULTIVATION OF THE ORDINARY PROTEUS BACILLUS, IN CONTACT WITH BLOOD AND BODY JUICES OF PATIENTS OR ANIMALS INFECTED WITH TYPHUS, MAKING THESE ORDINARY STRAINS AGGLUTINABLE, EXPLAINED THE REACTION. SUCH AN EXPERIMENT BY SILBER (287) IS QUOTED BY DAMMIN AND
BILLINGS (63). SILBER ENCLOSED PROTEUS BACILLI, WHICH WERE NOT AGGLUTINATED BY SERA FROM CASES OF TYPHUS, IN A COLLODION SAC. THIS SAC WAS PLACED INSIDE THE PERITOREAL CAVITY OF A GUINEA PIG SUFFERING FROM TYPHUS. THESE BACILLI BECAME AGGLUTINABLE AND THE ALTERED ANTIGENIC PROPERTIES REMAINED FOR MANY GENERATIONS.

WILSON (339) CONSIDERED THAT TYPHUS SERUM AGGLUTINATED MANY ORGANISMS OF WHICH PROTEUS X.19 IS ONE.

FELIX (84 & 85) HOWEVER, IN A FAIRLY EXHAUSTIVE SURVEY AND CRITICISM OF THE VARIOUS SEROLOGICAL EXPERIMENTS CARRIED OUT BY MANY OF THE EARLIER WORKERS INCLUDING WILSON, CONSIDERED THAT PROTEUS OX.19 IS QUITE SPECIFIC IN ITS PROPERTY OF BEING AGGLUTINATED BY TYPHUS SERA ONLY. HE PRODUCED EVIDENCE TO SHOW THAT THE AGGLUTINATING OF OTHER ORGANISMS, SUCH AS WILSON'S BACILLUS U2, BACILLUS AQUATALIS ALKALIGENES, AND BACILLUS COLI, MAY BE EXPLAINED IN VARIOUS WAYS. HE SUGGESTED THAT THE COMBINED ACTION OF AN ABUNDANCE OF PERFORMED NORMAL AGGLUTININS FOR SUCH SAPROPHYTES AS B. FAECALIS ALKALIGENES AND B. COLI, AND OF AUGMENTED SERUM COLLOIDS, WERE SOME OF THE FACTORS INVOLVED IN INCREASED HETEROLOGOUS AGGLUTININS (POLY-AGGLUTININS). HE SAID THAT B. PROTEUS X19 WAS NOT AFFECTED BY SUCH "POLY-AGGLUTINABLE" SERA.

CASTANEDA AND ZIA (45) WRITING IN 1933
CONSIDERED THAT THE RESULTS OF THEIR CROSS-
AGGLUTINATION AND ADSORPTION EXPERIMENTS FOLLOW
WITH CONSIDERABLE ACCURACY THE KNOWN CONDITIONS
FOR THE SO-CALLED MAJOR AND MINOR AGGLUTININS.
THEY ALSO CONSIDERED THAT THERE IS A COMMON ANTI-
GENIC FACTOR IN RICKETTSIA PROWAZEKI AND PROTEUS
X19. CASTANEDA'S (44) FINDING REPORTED IN 1936
OF HEAT STABILE EXTRACTS FROM RICKETTSIA
PROWAZEKI AND PROTEUS X19, WHICH CROSS-PRECIPIT-
ATED WITH BOTH ANTI-TYPHUS AND ANTIPROTEUS
SERUM, SUPPORTED THIS HYPOTHESIS. HE (44) HAS
ALSO DEMONSTRATED COMPLIMENT-BINDING ANTIBODIES
FOR RICKETTSIAE IN PROTEUS X19 ANTISERUM. TWO
UNEXPLAINED FACTS NOTED BY HIM ARE THAT TYPHUS-
IMMUNE SERUM POSSESSED NO COMPLIMENT-BINDING
ACTIVITY IN THE PRESENCE OF PROTEUS X19, WHICH
IS RATHER STRANGE AS THE REVERSE CROSS-REACTION
TOOK PLACE BETWEEN ANTIPROTEUS X19 SERUM AND
RICKETTSIAE. ALSO THERE WAS A LACK OF DETEC-
TABLE ANTIBODIES FOR BACILLUS PROTEUS X19 IN
THE SERUM OF TYPHUS-IMMUNE GUINEA PIGS.
DAMMIN AND BILLINGS (63) STUDYING THE WEIL
FELIX REACTION IN PATIENTS INFECTED WITH
BACILLUS PROTEUS AND PSEUDOMONAS AERUGINOSA
(B. PYOCYANEUS), FOUND FURTHER EVIDENCE OF A
COMMON MINOR ANTIGEN FROM CROSS-ADSORPTION TESTS.

THOUGH THE MECHANISM OF THE REACTION IS
STILL NOT COMPLETELY UNDERSTOOD, AND THE NATURE
OF THE ASSOCIATION BETWEEN PROTEUS X AND THE RICKETTSIAE IS COMPLETELY OBSCURE, NEVERTHELESS THE REACTION IS OF VERY CONSIDERABLE VALUE AS A DIAGNOSTIC AID. AS FELIX (87) POINTS OUT, "FOR THE PRESENT, MOST PATHOLOGISTS MUST CONTINUE TO RELY ON THE WEIL-FELIX AS THE SOLE TEST AVAILABLE FOR THE ROUTINE DIAGNOSIS OF TYPHUS FEVER". RESULTS MUST NATURALLY BE INTERPRETED BY CLINICIANS WITH DUE REGARD TO THE CLINICAL FINDINGS IN EACH CASE. THE TECHNIQUE MUST BE STANDARDIZED. FELIX AND OLITSKI (90) DISCUSS THE PRINCIPLES FULLY AND BRIDGES (30, 31 & 32) DESCRIBES THE PREPARATION OF SUSPENSIONS.

ACCORDING TO FELIX (87) THE OXK SUSPENSIONS ARE MORE SUSCEPTIBLE TO NON-SPECIFIC "NORMAL" AGGLUTININS. THEY ARE MORE DIFFICULT TO MAKE, AND THE ALCOHOL TREATED SUSPENSIONS OFTEN SHOW A CERTAIN DEGREE OF GRANULARITY WHICH INCREASES ON KEEPING. ALSO FELIX (89) STATES THAT UNDER UNSUITABLE CONDITIONS SUSPENSIONS DETERIORATE, OXK BEING THE FIRST TO DO SO.

THE LEVEL OF AGGLUTININS FOR PROTEUS X IN NORMAL HEALTHY INDIVIDUALS.

FELIX (87) CONSIDERS THAT AGGLUTININS PRESENT IN APPARENTLY NORMAL PEOPLE ARE RESIDUAL FROM A PREVIOUS INFECTION WITH RICKETTSIAE. THE
HEIGHT OF THE TITRE DEPENDS ON THE HEIGHT OF THE MAXIMUM TITRE DURING THE DISEASE. HE QUOTES WEIL'S (328) WORK ON 1837 CONTROLS IN 1916-18, AND CONCLUDES THAT TITRES OF OVER 1/100 ARE UNLIKELY TO BE ENCOUNTERED. BUT WHERE THERE IS A POSSIBILITY OF A RECENT PREVIOUS INFECTION, HIGHER TITRES MAY BE FOUND. FELIX (87) FINDS THAT PATIENTS WITH SUCH RAISED TITRES SHOW NO EVIDENCE OF NON-SPECIFIC RE-STIMULATION OF THESE AGGLUTININS IN THE COURSE OF TYPHOID, PNEUMONIA OR OTHER FEBRILE DISEASES.

SAVOOR, CASTANEDA AND ZINNSER (275) TESTED 600 CONTROLS IN THE UNITED STATES OF AMERICA. 0.6% HAD A TITRE OF 1/160. NO OTHERS WERE OVER 1/50.

BUCHWALD (38) FOUND NONE OVER 1/50 IN 300 PATIENTS SAID TO BE FREE FROM ANY SUSPICION OF TYPHUS.

IN INDIA AND BURMA CONTROLS ON "NORMALS" HAVE ONLY BEEN REPORTED BY TWO WORKERS. PHIPSON (245) EXAMINED 100 PERSONS, MAINLY NATIVES OF THE COUNTRY, DURING A SMALL OUTBREAK OF WHAT WAS PROBABLY LOUSE-BORNE TYPHUS IN SIMLA. NONE WERE POSITIVE OVER 1/8 TO LIVE CULTURES OF PROTEUS X.19. WHILE THESE RESULTS ARE NOT STRICTLY COMPARABLE WITH RESULTS CARRIED OUT BY THE PRESENT DAY TECHNIQUE, EVEN A CORRECTION FACTOR OF FOUR WOULD STILL GIVE A LOW TITRE.
In 1935 Scales, (277) using a more satisfactory technique, examined the Weil-Felix results in one hundred healthy Indians. In only one case were any suspensions agglutinated in a titre of over 1/50. The one case agglutinated Oxk to 1/125. A careful history offered no suggestion to explain the rise.

From the literature therefore, one may conclude that for the more completely investigated exanthematic typhus, titres of over 1/50 are rarely encountered in normal healthy individuals in Europe and the United States of America. In Malaya for the less completely investigated tsutsugamushi disease or scrub typhus, Lewthwaite and Savoor (155) accepted a titre of 1/125, obtained with alcoholized suspensions as diagnostic, provided the "tsutsugamushi syndrome" was present. This figure must be accepted with caution and with due consideration in any individual case.

The level of agglutinins for Proteus X in individuals with diseases other than typhus.

(1) Non-specific reactions.

Dick (70) in the Middle East, reported a considerable number of "non-specific" reactions, particularly to Oxk. As he had no reason to suppose that there could be any mite typhus in the area, he discontinued the use of Oxk.
TABLE ONE.
RASED WEIL-FELIX TITRES IN DISEASES OTHER THAN TYPHUS FROM DICK'S CASES SEEN IN THE MIDDLE EAST, AND CONSIDERED BY FELIX TO BE DUE TO DETERIORATION OF THE PROTEUS SUSPENSIONS.

<table>
<thead>
<tr>
<th>TOTAL NUMBER OF CASES</th>
<th>DISEASES</th>
<th>TITRE</th>
<th>NUMBER OF CASES SHOWING THE RISE IN TITRE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0X2</td>
<td>0X19</td>
</tr>
<tr>
<td>26</td>
<td>TYPHOID FEVER</td>
<td>(\frac{1}{120})</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(\frac{1}{240})</td>
<td>3</td>
</tr>
<tr>
<td>28</td>
<td>PARATYPHOID A &amp; B.</td>
<td>(\frac{1}{240})</td>
<td>8</td>
</tr>
<tr>
<td>168</td>
<td>OTHER UNSPECIFIED FEVERS.</td>
<td>(\frac{1}{120})</td>
<td>52</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(\frac{1}{240})</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(\frac{1}{480})</td>
<td>2</td>
</tr>
<tr>
<td>6</td>
<td>MEASLES</td>
<td>(\frac{1}{120})</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(\frac{1}{480})</td>
<td>1</td>
</tr>
<tr>
<td>1</td>
<td>COMMON COLD</td>
<td>(\frac{1}{480})</td>
<td>1</td>
</tr>
<tr>
<td>1</td>
<td>INFLUENZAL BRONCHO-PNEUMONIA</td>
<td>(\frac{1}{240})</td>
<td>1</td>
</tr>
<tr>
<td>1</td>
<td>INFECTIVE HEPATITIS</td>
<td>(\frac{1}{120})</td>
<td>1</td>
</tr>
<tr>
<td>SEVERAL</td>
<td>PSEUXIA OF UNKNOWN ORIGIN</td>
<td>(\frac{1}{250})</td>
<td>(\text{SEVERAL})</td>
</tr>
<tr>
<td>SEVERAL</td>
<td>SEPTIC CONDITIONS</td>
<td>(\frac{1}{120})</td>
<td>(\text{SEVERAL})</td>
</tr>
<tr>
<td>2</td>
<td>GLANDULAR FEVER</td>
<td>(\frac{1}{120})</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>BACILLARY DYSENTRY</td>
<td>(\frac{1}{120})</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>GASTRO-ENTERITIS</td>
<td>(\frac{1}{120})</td>
<td>2</td>
</tr>
<tr>
<td>1</td>
<td>LYMPHADENOMA</td>
<td>(\frac{1}{960})</td>
<td>1</td>
</tr>
</tbody>
</table>
SUSPENSIONS. MEGAW (198) QUOTES A PERSONAL COMMUNICATION FROM FELIX WHO CONSIDERS THAT THE STANDARD R.A.M.C. SUSPENSIONS USED BY DICK WERE UNRELIABLE. HE STATES THAT THE OXK SUSPENSIONS WERE FIRST TO DETERIORATE, OX2 NEXT, AND OX19 WERE RELATIVELY STABLE. MEGAW (198) ALSO REFER TO A PAPER BY VAN ROOYEN WHERE ANOMALOUS RESULTS WERE OBTAINED, AND FOR THE SAME REASON ACCORDING TO FELIX. FELIX (87) CONSIDERS THAT SOME OF VAN ROOYEN AND BEACROFT'S INCREASED TITRES OF OXK IN EPIDEMIC AND MURINE TYPHUS WERE DUE TO THIS NON-SPECIFIC REACTION. ONE WAS GLAD TO HAVE THE CONFIRMATION OF ONE'S OPINION BY SUCH AN AUTHORITY AS FELIX, THAT THE ANOMALOUS RESULTS REPORTED BY DICK (70) EVEN WHEN CARRIED OUT BY SUCH AN EMINENT WORKER AS VAN ROOYEN WERE DUE TO A NON-SPECIFIC FACTOR. A TABLE SHOWS DICK'S FINDINGS. SEE TABLE ONE.

(2) THE ENTERIC FEVERS.

AS MENTIONED BY FELIX (84) IT WAS EARLY RECOGNISED BY MANY OF THE CONTINENTAL WORKERS AND BY WEIL AND FELIX (329 & 330) THEMSELVES, THAT THERE WAS A NON-SPECIFIC STIMULATION BY TYPHUS (AND OTHER FEBRILE DISEASES), OF TYPHOID AGGLUTININS IN PATIENTS PREVIOUSLY INOCULATED BY A TYPHOID VACCINE, OR WHO HAD PREVIOUSLY HAD ENTERIC FEVER. ON THE OTHER HAND AGGLUTININS FROM PROTEUS X19 ARE NOT STIMULATED BY INTERCURRENT INFECTIONS. I DO NOT INTEND TO DISCUSS
THE CONSIDERABLE LITERATURE. FELIX'S (87) OPINION WOULD APPEAR TO BE THAT AS A RAISED TITRE TO PROTEUS X MAY PERSIST FOR MONTHS OR YEARS, ITS DURATION DEPENDING ON ITS ORIGINAL HEIGHT, ANY RAISED TITRE TO OX19 IS DUE TO TYPHUS, PRESENT OR PAST, FRANK OR 'INAPARENT'. THE POSITION WITH REGARD TO THE LESS FULLY EXPLORED PROTEUS OXK IN THE PRESENCE OF OTHER DISEASES IS NOT QUITE SO CLEAR. IN INDIA, SCALES (277) FOUND NO RISE OF PROTEUS AGGLUTININS FOR OX2, OX19 OR OXK IN THREE CASES OF PARATYPHOID A FEVER, AND FIVE CASES OF TYPHOID FEVER, ALL BACTERIOLOGICALLY PROV ED.

(3) BRUCELLIASIS.

NICOLLE AND COMTE (225) IN 1910 HAD REPORTED AGGLUTINATION OF BRUCELLA MELITENSIS BY TYPHUS SERUM. THOSE AGGLUTININS DISAPPEARED IN TWO DAYS WHEN PROTEUS AGGLUTININS DEVELOPED. FELIX (85) HOWEVER, REPORTS THAT THOUGH HE AND OTHER WORKERS WERE ABLE TO CONFIRM THIS FINDING, POINTS OUT THAT THE AGGLUTININS RECORDED BY NICOLLE AND COMTE WERE WITHIN THE LIMITS SEEN IN APPARENTLY NORMAL PEOPLE.

CALDER (43) QUOTES KEMP, ET AL (134), WHO IN 1933 REPORTED IN THE TEXAS STATE JOURNAL OF MEDICINE FOUR CASES OF UNDULANT FEVER IN WHICH WERE FOUND PROTEUS AGGLUTININS TO TITRES OF 1/80-1/240. THERE WAS HOWEVER, NO INCREASE IN TITRE AS THE DISEASE PROGRESSED. LEE FOSHAY'S (96)
ARTICLE ON THE LABORATORY DIAGNOSIS OF UNDULANT FEVER DEMONSTRATES THAT IN CERTAIN AREAS OF THE UNITED STATES, DIAGNOSTICIANS ARE PRONE TO USE THE LABEL OF "CHRONIC BRUCELLOSIS" FOR CHRONIC PYREXIAS ON TENUOUS GROUNDS. CALDER (43) HAS SELECTED CASES PROBABLY SUFFERING FROM CHRONIC BRUCELLOSIS AND THE CONTROLS WITH GREAT CARE. 13.5% AGGLUTINATED PROTEUS X19 IN DILUTIONS OF 1/80 OR OVER. A FOLLOW UP SHOWED NO SIGNIFICANT CHANGE IN PROTEUS OR BRUCELLA AGGLUTININS. CALDER CONSIDERS PROTEUS A MORE EASILY AGGLUTINABLE ANTIGEN THAN BRUCELLA, AND BRUCELLA AGGLUTININS ARE OFTEN LOW OR ABSENT IN CHRONIC BRUCELLOSIS. HE ALSO CONSIDERS ANTI-BRUCELLA SERA UNRELIABLE FOR DIAGNOSIS OWING TO ITS POLYAGGLUTINATING POWER. LEE FOSHAY (96) CONSIDERS THAT BOTH PROTEUS X19 AND BRUCELLA HAVE A VERY PROTEAN ANTIGENIC NATURE. AGGLUTININS FOR PASTURELLA TULARENSIS WERE SELDOM ENCOUNTERED AND WHEN PRESENT WERE IN LOW TITRE AND WERE NOT LIKELY TO LEAD TO CONFUSION IN CHRONICALLY ILL PATIENTS. CALDER DISCUSSES THE POSSIBILITY OF RICKETTSIAL DISEASE OR A PROTEUS INFECTION BEING RESPONSIBLE FOR THE RISE IN PROTEUS AGGLUTININS. AS PROTEUS INFECTION IS NOT KNOWN TO PRODUCE THAT DISEASE PICTURE, AND AS FAR AS IS KNOWN THERE IS NO CHRONIC RICKETTSIOSIS, HE EXCLUDES THESE TWO POSSIBLE EXPLANATIONS OF THE RISE IN PROTEUS
AGGLUTININS. There was no previous history to suggest infection which might have produced a reaction of an amnestic nature. Calder concludes that the antigenic relationship between Proteus X and Brucella is responsible for the confused serological findings. He suggests that if agglutinins for Proteus X19 are found in chronically ill patients in the United States, and if the titre does not rise, Bruceliasis (Brucellosis) should be suspected.

I have not encountered a case of Bruceliasis in Burma and India during my twenty years practice. I have not read of any cases where there has been confusion. If Calder's (43) results are accepted, a constant titre to Proteus X19 in an area where Brucellosis is endemic is consistent with a Brucella infection.

(4) TOXOPLASMOSIS.

The early reports that in certain cases of fever a Toxoplasma was pathogenic were received with scepticism. Weynon (332) considered some of the descriptions of the parasite were of artefacts. Wolf and Cowan (344) however, reported seven cases of a congenital granulomatous encephalomyelitis in newly born children, in which a Toxoplasma was established as the cause. Sabin (269) reported two cases in children.
Pinkerton and Henderson (250) report two exceedingly interesting cases of toxoplasmosis in adults. Both patients had been bitten by ticks which seems likely to have been the vector. The onset was sudden; an eruption resembling American typhus was present; there was pulmonary involvement in both cases. Proteus X19 was agglutinated in one case to 1/160.

The morbid histology of the lung lesions was very like a typical pneumonia of unknown etiology. The protozoon was present in scanty numbers, except that when found, very many were seen in a single cell. Injection of blood intraperitoneally into guinea pigs produced a febrile reaction like typhus and histologically small collections of round cells in the brain. Findlay (94) points out that until recently the "nodules" seen in a guinea pig's brain following intraperitoneal injection of blood were thought to be specific for typhus. In the case of toxoplasmosis however, the parasites may be found in the peritoneal exudate. The disease has not been described in human beings in Britain, and as far as we know at present is exceedingly rare. But we are not in a position to say with absolute certainty that it does not exist in India or Burma. It must be kept in mind until further information is available.
(5) LEPTOSPIROSIS.

Lewthwaite and Savoor (155) noted a constant titre of 1/480 to Proteus OXK in two out of eleven cases of leptospirosis. They consider that the disease can be differentiated from typhus clinically. It can be reproduced in guinea pigs. Leptospirosis is being reported in increasing numbers from various parts of the world. India is a country where such a disease might flourish and it should be borne in mind. I have never seen a case, though I have on several occasions suspected it to be present.

(6) RELAPSING FEVER.

Elsdon-Dew (77) reports OXK agglutinins to a titre of 1/100 in relapsing fever, in six out of thirty-five cases with no relapses, but in twenty-two out of twenty-seven cases with one or more relapses. In some relapsing cases the titre rose to 1/6400, falling within a month after the last relapse to a titre of 1/80.

(7) RAT BITE FEVER.

This infection was found by Lewthwaite and Savoor (156) to produce agglutinins for Proteus OXK in rabbits. Experimental infection of the three tabetic patients and four Macaeus monkeys produced no increase in OXK agglutinins in two of the men and two of the monkeys. In the third man and the other two monkeys there was
A slight rise, which by their standards would have been considered negative. I have not seen a case of rat bite fever. From descriptions the bite is distinctive.

(8) Suppuration due to Bacillus Proteus.

This is a relatively uncommon infection, usually associated with other organisms. Sonnenschein (293) has described two cases of infection with Bacillus Proteus vulgaris and a streptococcus in which agglutinins for Ox19 were present in titres of 1/1600 and 1/200 respectively. Agglutinins for the patients' Proteus were absent. Sonnenschein therefore suggests that a titre of less than 1/2000 is inconclusive in febrile patients with sepsis, unless the symptoms point definitely to typhus. It is not evident from the summary whether the possibility of a previous attack of typhus had been excluded.

Felix (87) has pointed out that depending on height of the titre during the disease, residual agglutinins may be present for a very long time. Sonnenschein found that rickettsial suspensions were also agglutinated. Residual rickettsial agglutinins are also present for a long time.

Damin and Billings (63) were able to report fourteen cases, mainly urinary infections, where Proteus organisms were isolated. It was not assumed that these organisms necessarily
PLAYED A MAJOR ROLE IN THE PATIENTS' DISEASE. TWELVE WERE PROTEUS MIRABALIS AND TWO PROTEUS VULGARIS. IN NINE OF THESE CASES A TITRE OF 1/160 WAS FOUND FOR PROTEUS OX AGGLUTININS. IN SEVEN CASES TITRES OF 1/640 OR OVER WERERecorded. IT WAS FOUND THAT ONLY THOSE WHO DEVELOPED APPRECIABLE AGGLUTINATION TITRES TO THEIR OWN PROTEUS, AND PARTICULARLY TO THE 'O' ANTIGEN, SHOWED AGGLUTINATION IN SIGNIFICANT TITRE FOR PROTEUS OX ANTIGENS. CROSS-AGGLUTINATION TESTS SUGGESTED A COMMON MINOR ANTIGEN FOR PROTEUS X AND THE PATIENTS' PROTEUS ORGANISMS. DAMMIN AND BILLINGS QUOTE DUTCH WORKERS IN THE EAST INDIES WHO FOUND A SIMILAR PATTERN IN FIFTY-TWO STRAINS, AND REFER TO SILBER'S (287) WORK WHICH HAS ALREADY BEEN MENTIONED. ADSORPTION EXPERIMENTS BY DAMMIN AND BILLINGS SUGGEST THAT THE PROTEUS ORGANISMS ISOLATED FROM THEIR CASES WERE ANTIGENICALLY MORE CLOSELY RELATED TO OXK, THAN TO OX2 AND OX19. ONE MAY CONCLUDE THEREFORE, THAT THE FINDINGS OF A RAISED TITRE OF AGGLUTININS TO PROTEUS OX IN ANY FEBRILE CASE WITH SEPSIS OR URINARY INFECTION MUST BE REGARDED WITH SUSPICION, PARTICULARLY IF THERE IS A SIGNIFICANT RISE IN THE OXK. WOLFF (341) QUOTES PRAUSNITZ (257) WHO RECORDED THE OBSERVATION THAT THE HEATING OF SERUM DESTROYS X AGGLUTININS FOR PROTEUS VULGARIS. THIS FACT IS WELL KNOWN AND IS
MENTIONED BY FELIX (87). YACOB (348) HAS INVESTIGATED THE RELATIONSHIP BETWEEN THE PROTEUS 'X' GROUP OF STRAINS ASSOCIATED WITH TYPHUS AND THE PROTEUS BACILLUS DERIVED FROM OTHER SOURCES, AND CONFIRMS THIS FINDING. FELIX (85) ADVISES HEATING SERA OF POSSIBLE TYPHUS CASES FOR HALF AN HOUR TO 45° C. THIS DESTROYS A HEAT LABILE RICKETTSIAL ANTIGEN AND PREVENTS IT INHIBITING THE INTERACTION BETWEEN THE HEAT-STABLE ANTIGEN AND ITS CORRESPONDING ANTIBODY.

(9) MISCELLANEOUS CONDITIONS. MALARIA, KALA-AZAR, SPLEENOMEGALY, MISCELLANEOUS FEBRILE CONDITIONS, MISCELLANEOUS AFEBRILE CONDITIONS INCLUDING ANAEMIA.

IN CALCUTTA IN 1936 PASRICHA, PANJA AND LAL (241) EXAMINED SERA FROM TWO HUNDRED AND EIGHTY ADMISSIONS TO THE CARMICHAEL HOSPITAL FOR TROPICAL DISEASES SUFFERING FROM THE ABOVE CONDITIONS IN 1935. NONE WERE CONSIDERED TO BE SUFFERING FROM TYPHOID OR PARATYPHOID FEVER. NONE HAD BEEN INOCULATED AGAINST THE TYPHOID-PARATYPHOID GROUP. WIDAL RESULTS SUGGESTED THAT INFECTION WITH THOSE ORGANISMS WAS COMMON IN THE POPULATION OF WHICH THESE PATIENTS WERE A CROSS SAMPLE. WEIL-FELIX TESTS WERE CARRIED OUT ON THE SAME SERA (242). THE FIGURES IN THE TWO ARTICLES ARE NOT QUITE CONSISTENT. 1.4% OF 280 CASES WERE POSITIVE TO OX19 IN 1/100 AND OVER. NONE WERE POSITIVE TO OX2 IN 1/100 OF 259 CASES. OF 77 CASES 7.8% WERE
POSITIVE TO OXK IN DILUTIONS OF 1/100 OR OVER.
IT IS NOT POSSIBLE FROM THESE TABLES TO TELL IN WHICH CONDITIONS THESE READINGS WERE FOUND, AS THE FINDINGS IN THE VARIOUS CLINICAL CONDITIONS ARE GROUPED AS POSITIVE IN DILUTIONS OF ONLY 1/25 OR OVER. THE POSSIBILITY OF PREVIOUS INFECTION WITH TYPHUS, FRANK OR 'INAPPARENTE' CANNOT BE EXCLUDED.

THE PROTEUS SUSPENSIONS WERE PREPARED IN THE BACTERIOLOGICAL DEPARTMENT OF THE SCHOOL OF TROPICAL MEDICINE, CALCUTTA.

PERSONAL FINDINGS IN INDIA OF WEIL-FELIX TITRES IN DISEASES OTHER THAN TYPHUS.

(1) ENTERIC FEVER.
DURING THREE AND THREE QUARTER YEARS WHILE IN THE ARMY IN INDIA I SAW TWENTY CASES OF ENTERIC FEVER BACTERIOLOGICALLY PROVED. ALL BACTERIOLOGICAL FINDINGS WERE CONFIRMED BY THE ARMY ENTERIC LABORATORY IN KASAULI.

(IA) TYPHOID FEVER.
I SAW ONLY TWO BACTERIOLOGICALLY PROVED CASES OF TYPHOID FEVER, AND FIGURES ARE NOT AVAILABLE FOR ONE OF THESE. THE OTHER, AN INDIAN, HAD NOT HAD T.A.B. INOCULATIONS. HIS WEIL-FELIX TITRE WAS NOT RAISED.

SEE TABLE TWO.

(IB) PARATYPHOID A FEVER.
FOUR CASES WERE SEEN. THREE HAD A TIRE
### Table Two.

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<th>Day of Disease</th>
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</tr>
<tr>
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<td>1/25</td>
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### Table Three.

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<th>Day of Disease</th>
<th>Titre of Weil-Felix</th>
<th>8</th>
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<th>19</th>
<th>24</th>
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<td>1/50</td>
<td>1/25</td>
<td>1/25</td>
<td></td>
<td>0</td>
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<td></td>
</tr>
<tr>
<td>Ox19</td>
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<td>1/25</td>
<td>1/25</td>
<td></td>
<td>0</td>
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<td>1/25</td>
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<td></td>
<td></td>
</tr>
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<tr>
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<td>1/50</td>
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</table>
OF 1/50 TO ALL THREE PROTEUS SUSPENSIONS AT ONE
TIME OR ANOTHER. ONE CASE AGGLUTINATED OX19
ON THE EIGHTH DAY TO 1/125, THE TITRE FALLING
THEREAFTER. ALL HAD HAD PROTECTIVE T.A.B.
INOCULATIONS.

SEE TABLE THREE.

(1c) PARATYPHOID B FEVER.

IN TWO CASES OF PARATYPHOID B FEVER, ON
THE TWELFTH AND NINETEENTH DAYS, AND ON THE
FIFTEENTH AND TWENTY-FIFTH DAYS RESPECTIVELY,
THERE WAS NO RISE IN THE TITRE FOR OX2, OX19
OR OXK.

(1d) PARATYPHOID C FEVER.

FIVE CASES OF PARATYPHOID C FEVER WERE
SEEN, FOUR BACTERIOLOGICALLY PROVED BY BLOOD
CULTURE, AND ONE BACTERIOLOGICALLY PROVED BY
STOOL CULTURE. IN ONE CASE OX19 AGGLUTININS
WERE PRESENT TO A TITRE OF 1/250. IN TWO
CASES OXK AGGLUTININS WERE PRESENT TO TITRES
OF 1/125 AND 1/250 RESPECTIVELY. THESE CASES
CLINICALLY MIGHT BE CONFUSED WITH TYPHUS.
THE CASE WITH A TITRE OF 1/250 OXK DIED. THE
POST MORTEM MORBID ANATOMY WAS CONSISTENT WITH
AN ACUTE TOXAEMIA, BUT HAD FEATURES NOT ENTIRELY
UNLIKE TYPHUS. ONE MIGHT CONSIDER, THOUGH I
THINK IT UNLIKELY, THAT HE MAY HAVE HAD A
DOUBLE INFECTION.

SEE TABLE FOUR.
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**Note:** A FATAL CASE
SALMONELLA ENTERITIDIS GAERTNER INFECTIONS.

SIX CASES OF ENTERIC FEVER IN WHICH SALMONELLA ENTERITIDIS (B. ENTERITIDIS) GAERTNER WAS ISOLATED FROM THE BLOOD, AND ONE CASE IN WHICH THE SAME ORGANISM WAS GROWN FROM THE BILE AT AUTOPSY ARE RECORDED HERE. IN TWO CASES THE TITRE OF PROTEUS AGGLUTININS WAS 1/125, OXK AND OX2 RESPECTIVELY. CLINICALLY THESE CASES MIGHT ALSO BE CONFUSED WITH SCRUB TYPHUS.

SEE TABLE FIVE.

THESE RESULTS FROM ENTERIC FEVER CASES SEEN BY MYSELF ARE NUMERICALLY SMALL. THEY DO SHOW HOWEVER, THAT TITRES OF 1/125 OR EVEN 1/250 ARE NOT IN THEMSELVES DIAGNOSTIC OF TYPHUS OR TYPHUS-LIKE FEVER. IT IS OF COURSE DIFFICULT TO EXPLAIN THE RISE IN TITRE SEEN ON OCCASIONS. IN NONE OF THESE CASES COULD A HISTORY SUGGESTIVE OF A RECENT ATTACK OF TYPHUS LIKE FEVER BE OBTAINED. SUCH AN ATTACK MIGHT GIVE A DELAYED RISE IN PROTEUS AGGLUTININS. FAULTS IN TECHNIQUE COULD I THINK BE EXCLUDED, AS MY COLLEAGUE IN THE CALCUTTA DISTRICT LABORATORY, MAJOR PARKER R.A.M.C., AN EXPERIENCED BACTERIOLOGIST, WAS FULLY AWARE OF THE NECESSITY OF BEING ON THE LOOK OUT FOR PITFALLS. DETERIORATION OF SUSPENSIONS MIGHT BE CONSIDERED TO BE A DISTINCT POSSIBILITY, AND I CANNOT EXCLUDE THAT POSSIBILITY, EXCEPT TO SAY THAT FALSE POSITIVES WERE NOT NOTED.
<table>
<thead>
<tr>
<th>TABLE FIVE.</th>
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<tbody>
<tr>
<td>TITRE OF AGGLUTININS IN SEVEN CASES OF PARATYPHOID (SALMONELLA ENTERITIDIS GÄERTNER) FEVER.</td>
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<td>O</td>
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O: Negative
1/25: Moderate positive
1/50: Strong positive
1/25: High positive
IN THE NUMEROUS CASES OF BRONCHITIS, AND SUSPECTED TYPHUS OR ENTERIC FEVER WHICH WERE CARRIED OUT IN THE SAME LABORATORY BY MAJOR PARKER TO WHICH I WAS ATTACHED, FOR A BRITISH GENERAL HOSPITAL AS WELL AS OTHER MEDICAL UNITS IN GREATER CALCUTTA. SUSPENSIONS WERE FLOWN FROM KASAULI, AND WERE FREQUENTLY RENEWED.

(2) PYREXIA OF UNKNOWN ORIGIN.

THREE CASES OF FEVER FOR WHICH NO OBVIOUS CAUSE COULD BE FOUND, AND FROM THE BLOOD OF WHICH BACILLUS FAECAIIS ALKALIGENES WAS ISOLATED, DID NOT SHOW ANY RISE IN TITRE OF PROTEUS AGGLUTININS.

SEE TABLE SIX.

(3) KALA AZAR.

IN TWO CASES OF THIS DISEASE A RISE OF OXK AGGLUTININS WAS SEEN. ONE CASE RESISTED DIAGNOSIS UNTIL THE 124TH DAY OF FEVER. AFTER SEVERAL NEGATIVE STERNAL PUNCTURES, LEISHMAN DONOVAN BODIES WERE FOUND IN SPLENIC SMEARS. THE OXK AGGLUTININS ROSE TO 1/125 ON THE 61ST DAY OF FEVER, FALLING THEREAFTER. THE PATIENT HAD BEEN IN HOSPITAL FOR 23 DAYS PRIOR TO THE FIRST WEIL-FELIX TEST. THERE WAS NO APPARENT DETERIORATION IN HIS CONDITION TO SUGGEST THAT HE WAS SUFFERING FROM AN ACUTE INFECTION IN ADDITION TO KALA-AZAR. A SECOND CASE WAS MORE SUGGESTIVE CLINICALLY BUT THERE WAS NO RISE OR
### Table Six.

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</tbody>
</table>
FALL IN AGGLUTININ TITRE OVER TEN DAYS. THIS FEATURE OF COURSE PRECLUDES DIAGNOSIS. BOTH CASES MADE AN UNINTERRUPTED RECOVERY AFTER THE ADMINISTRATION OF UREA-STIBAMINE.

SEE TABLE SEVEN.

I HAVE SEEN ABOUT THIRTY-FIVE OTHER CASES OF KALA-AZAR. THE WEIL-FELIX TEST WAS CARRIED OUT IN TWENTY OF THESE DURING ROUTINE INVESTIGATIONS. IN NONE OF THESE TWENTY CASES WAS THE TITRE AT ANY TIME SIGNIFICANT. SEN GUPTA (282) IN 1944 REPORTED A CASE WHERE HE CONSIDERED THAT TYPHUS FEVER COMPLICATED KALA-AZAR. THE DIAGNOSIS WAS MADE FROM A SUGGESTIVE FEVER, A RASH AND A TITRE OF 1/400 OXK FALLING TO 1/25 FOUR DAYS LATER. IT MAY BE POSSIBLE THAT RAISED SERUM PROTEIN MAY INFLUENCE THE REACTION OCCASIONALLY, THOUGH FELIX (85) HAS STATED THAT OX19 IS NOT AGGLUTINATED IN DISEASES KNOWN TO PRODUCE AN INCREASE IN SERUM GLOBULIN. FURTHER INVESTIGATION IS REQUIRED IN KALA-AZAR. MEANWHILE, IN CASES WHERE THIS DISEASE IS A POSSIBILITY, THE WEIL-FELIX SHOULD BE CRITICALLY INTERPRETED.

THE VALUE OF THE WEIL-FELIX TEST IN THE DIAGNOSIS OF TYPHUS AND TYPHUS-LIKE FEVERS.

THIS TEST, IN SPITE OF ITS ANOMALIES, REMAINS AN INVALUABLE ANCILLARY AID TO DIAGNOSIS. A DISEASE MAY BE SUSPECTED, BUT RARELY DIAGNOSED IN THE LABORATORY, PARTICULARLY ON THE RESULT OF
ONE TEST. AN EXAMPLE MAY BE QUOTED FOR THE WEIL-FELIX TEST. A FOLLOWER, A 'DHOBIE' CR WASHERMAN, WAS ADMITTED WITH A COMPLAINT OF HAEMOPTYSIS.

HE WAS ATTACHED TO A UNIT FROM WHICH WE HAD BEEN SENT MANY CASES OF SCRUB TYPHUS. FOR NINE DAYS HIS TEMPERATURE NEVER ROSE ABOVE 99°. FOR THE NEXT FOUR DAYS HE HAD FEVER UP TO 103° - THEREAFTER HE BECAME NORMAL. HE GAVE A HISTORY OF HAVING HAD FEVER WITH RIGORS EVERY OTHER DAY DURING THE PREVIOUS THREE MONTHS. DURING THAT TIME HE WAS TREATED AS AN OUT-PATIENT IN A FIELD AMBULANCE. ON THE 14TH DAY AFTER ADMISSION THE OXK AGGLUTININS WERE PRESENT TO A TITRE OF 1/2500 - GRADUALLY FALLING DURING THE NEXT 35 DAYS. IT WOULD SEEM REASONABLE TO ASSUME THAT HE HAD AN ATTACK OF SCRUB TYPHUS PRIOR TO ADMISSION, FOR WHICH RESIDUAL AGGLUTININS REMAINED. HIS FEVER WHILE IN HOSPITAL WAS NOT TYPHUS.

SEE TABLE EIGHT.

IN EXANTHEMATIC TYPHUS ONE MAY EXPECT A RISE IN TITRE AT THE END OF THE FIRST WEEK; IN TSUTSUGAMUSHI DISEASE (SCRUB TYPHUS) ONE DOES NOT EXPECT TO SEE IT UNTIL LATER, USUALLY IN THE SECOND WEEK, BUT SOMETIMES NOT UNTIL CONVALESCENCE HAS SET IN. TO BE OF VALUE SERIAL TESTS ARE NECESSARY TO SHOW THE WAXING AND WANING OF AGGLUTININS.
TABLE EIGHT

TEMPERATURE CHART AND SERIAL WEIL-FELIX RESULTS IN A CASE WITH A HISTORY OF UNDIAGNOSED FEVER DURING THE THREE MONTHS PRIOR TO ADMISSION TO HOSPITAL, AND ADMITTED TO HOSPITAL DURING HIS CONVALESCENCE ON ACCOUNT OF HAEMOPTYSIS.

[Diagram showing temperature chart with serial Weil-Felix results during hospitalization]
IN FELIX'S (87) OPINION, A RISE OF ONE HUNDRED PER CENT IN TITRE IN CASES CLINICALLY SUGGESTIVE OF TYPHUS IS SIGNIFICANT. A TITRE OF 1/80 OR 1/100 IN AN UNVACCINATED PATIENT WHO DOES NOT RESIDE IN AN ENDEMIC AREA IS ALSO SIGNIFICANT. IF VACCINATED, 1/200 IS SUGGESTIVE. IF IN AN ENDEMIC AREA TITRES OF 1/200 ARE NOT SIGNIFICANT, BUT A RISING TITRE IS CONCLUSIVE; AN UNALTERED TITRE IS NEGATIVE, EXCEPT IN EXTREMELY SEVERE AND USUALLY FATAL CASES OF LOUSE TYPHUS.

OX19 AGGLUTININS MAY BE PRESENT IN BRUCELLIASIS, TOXOPLASMOSIS AND SUPPURATIVE PROCESSES DUE TO, OR ASSOCIATED WITH, BACILLUS PROTEUS, USUALLY IN BORDER LINE TITRES AND NOT EXHIBITING WAXING AND WANING OF TITRE. BORDER LINE TITRES FOR OX19 MAY OCCUR IN PARATYPHOID A OR C. THE EXPLANATION IS NOT AT PRESENT OBVIOUS.

DICK'S (70) RESULTS WHICH INCLUDE A CONSIDERABLE RISE IN TITRE TO OXK IN A CASE OF LYMPHADENOMA MAY BE LEGITIMATELY CONSIDERED TO BE DUE TO DETERIORATION IN THE SUSPENSIONS AS SUGGESTED BY FELIX (89).

OXK AGGLUTININS ARE REPORTED IN RAISED TITRE IN RELAPSING FEVER, PARTICULARLY WITH ONE OR MORE RELAPSES AND IN LEPTOSPIROSIS, BUT EXHIBIT NO WAXING AND WANING OF TITRE IN THE LATTER DISEASE. BORDER LINE TITRES OF OXK HAVE BEEN SEEN IN PARATYPHOID C, SALMONELLA ENTERITIDIS GAERTNER,
RAT BITE FEVER AND KALA AZAR.

THE CONDITIONS WITH BORDER LINE TITRES REQUIRE FURTHER INVESTIGATION. FALLACIES IN THE TEST INCLUDE FAULTY TECHNIQUE IN PREPARING THE SUSPENSIONS AND CARRYING OUT THE TEST, AND DETERIORATION OF SUSPENSIONS, PARTICULARLY OXK. FELIX AND RHODES (91) IN 1931 POINTED OUT THAT THE MOST IMPORTANT SOURCE OF ERROR IS H AGGLUTINATION WITH B. PROTEUS X STRAINS.

FINDLAY (94) SUGGESTS THAT IN CARRYING OUT THE WEILL-FELIX TEST, KNOWN NEGATIVE SERUM SHOULD BE INCLUDED TO EXCLUDE SPONTANEOUS AGGLUTINATION.

SEE TABLE NINE.

REMARKS ON THE CLASSIFICATION OF THE TYPHUS GROUP OF FEVERS.

THERE IS NO WHOLLY SATISFACTORY CLASSIFICATION OF THE TYPHUS GROUP OF FEVERS.

MEGAW'S (187) PROVISIONAL CLASSIFICATION BY VECTOR WAS A FIRST STEP AND A MOST USEFUL ONE. IN SO MANY CASES OF TYPHUS LIKE FEVER HOWEVER, IT IS IMPOSSIBLE TO COME TO ANY CONCLUSION ABOUT THE VECTOR OR POSSIBLE VECTOR. THE UNSATISFACTORY GROUP OF "VECTOR UNKNOWN" IS ALMOST EQUALLY BALANCED BY THE GROUP CLASSIFIED BY THEIR SEROLOGICAL RESPONSE TO THE WEILL-FELIX TEST AS "INDETERMINATE". CLASSIFICATION BY THE CAUSAL RICKETTSIA IS OUT OF THE QUESTION.
TABLE NINE.

DISEASES OTHER THAN TYPHUS LIKE FEVERS IN WHICH A COINCIDENT RAISED TITRE OF PROTEUS OX AGGLUTININS HAVE BEEN SEEN.

<table>
<thead>
<tr>
<th>IN LOW TITRE</th>
<th>OX2</th>
<th>OX19</th>
<th>OXK</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>BRUCELLIASIS.</td>
<td>LEPTOSPIROSIS.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TOXOPLASMOSIS</td>
<td>RAT-BITE FEVER.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>PROTEUS SUPPURATION.</td>
<td></td>
</tr>
</tbody>
</table>

| IN HIGH TITRE |                  | RELAPSING FEVER. (DICK'S CASE OF LYMPHADENOMA IS CONSIDERED TO BE A FALSE POSITIVE.) |
|---------------|------------------------------------------------|

<table>
<thead>
<tr>
<th>IN LOW TITRE</th>
<th>OX2</th>
<th>OX19</th>
<th>OXK</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B. ENTERITIDIS GAERTNER INFECTION.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>? KALA- AZAR</td>
<td>PARATYPHOID A FEVER.</td>
<td>B. ENTERITIDIS GAERTNER INFECTION.</td>
</tr>
<tr>
<td></td>
<td>? KALA- AZAR</td>
<td>PARATYPHOID C FEVER.</td>
<td>? KALA- AZAR.</td>
</tr>
</tbody>
</table>

PERSONAL FINDINGS
MEANTIME, EVEN IF IT SHOULD BE WHOLLY SATISFACTORY. THE ADOPTION OF A LOCAL NAME FOR A GROUP IS, AS MEGAW ORIGINALLY POINTED OUT, APT TO BE CONFUSING. SUCH QUALIFICATIONS AS 'EPIDEMIC' AND 'ENDEMIC' TYPHUS CAN BE MISLEADING. EPIDEMIC USUALLY REFERS TO CLASSICAL EXANTHEMATIC TYPHUS. THAT DISEASE CAN, ACCORDING TO FRIEDMAN, (99) BE ENDEMIC. ENDEMIC TYPHUS VERY FREQUENTLY REFERS TO MURINE TYPHUS, BUT IN THE UNITED STATES OF AMERICA IT HAS ALSO BEEN USED BY BADGER, DYER AND RUMREICH, (11) TO REFER TO ROCKY MOUNTAIN SPOTTED FEVER.

FELIX'S (86 & 87) CLASSIFICATION BY THE SEROLOGICAL RESPONSE TO PROTEUS OX ANTIGENS, IS PERHAPS THE ONLY MEANS BY WHICH A COMMON APPLICABLE TEST TO SOME EXTENT SORTS OUT ALL THE KNOWN TYPHUS AND TYPHUS-LIKE FEVERS. IT IS TRUE THAT THE TEST DOES NOT MAKE A DISTINCTION BETWEEN EXANTHEMATIC AND MURINE TYPHUS. ALSO, AS IN THE CLASSIFICATION BY VECTOR, THERE IS A GROUP OF NOT CLEARLY DIFFERENTIATED AS "TYPE UNDETERMINED", IN WHICH IS GROUPED ROCKY MOUNTAIN SPOTTED FEVER.

THERE ARE CLEARLY THREE MAIN GROUPS CORRESPONDING TO THE THREE KNOWN SPECIES OF RICKETTSIAE. FIRST THERE IS THE EXANTHEMATIC TYPHUS WITH ITS VARIATION, MURINE TYPHUS.
SECOND THERE IS TSUTSUGAMUSHI DISEASE. THIRD THERE IS ROCKY MOUNTAIN SPOTTED FEVER. IT REMAINS TO BE SEEN WHETHER THERE ARE ANY FURTHER SPECIES OR WHETHER THE AT PRESENT UNDETERMINED GROUP WILL BE FOUND TO BE MADE UP OF VARIANTS OF THE THREE MAIN GROUPS. AS ZINSSER AND BAYNE-JONES (356) REMARK, "IN THE TYPHUS, SPOTTED FEVER DIVISION, A NUMBER OF SUBVARIETIES WHICH SHADE INTO EACH OTHER IN SUCH A GRADUAL MANNER THAT A DEVELOPMENT OF ONE TYPE OF DISEASE FROM ANOTHER, BY ADAPTATION OF THE VIRUS TO DIFFERENT INSECT AND ANIMAL HOSTS, SUGGESTS ITSELF". THE CLASSIFICATION ON FELIX'S SEROLOGICAL SCHEME MAY BE CONVENIENTLY CONDENSED IN A TABLE.

SEE TABLE TEN.

THE LITERATURE OF TYPHUS IN INDIA.

# Table Ten

<table>
<thead>
<tr>
<th>General Distribution</th>
<th>Known Group</th>
<th>Known Causative Agent</th>
<th>Known Vector</th>
<th>Known Ticks</th>
<th>Known Louse Diseases</th>
<th>Known Rat Flea Diseases</th>
<th>Known Larval Mite Diseases</th>
<th>Known Tick Diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Russia, Poland, Balkans, Ireland, Eastern United States of America</td>
<td>Epidemic Typhus, European Typhus, Classical Typhus, Louse Typhus</td>
<td>Rickettsia prowazekii</td>
<td>Louse</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>World Wide, in Ports, Malaya, Simla</td>
<td>Endemic Typhus, Homen Disease, Fièvre Nauitique, Fièvre Endemic Benin, Flea Typhus</td>
<td></td>
<td>Rat Flea</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Japan, Formosa, Sumatra, Malaya, Burma, Bengal, Assam</td>
<td>Scrub Typhus, Pseudo-Typhus of Dell, Mossman Fever, Flood Fever, Mite Fever</td>
<td></td>
<td>Larval Mite</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>United States of America, Mediterranean Littoral, Brazil</td>
<td>&quot;Endemic&quot; Typhus, Spotted Fever, Fièvre Boutonneuse, Sao Paulo Typhus, Tick Typhus</td>
<td></td>
<td>Tick</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Causal Vector**

- Rickettsia prowazekii
- Rickettsia orientalis
- Rickettsia rickettsii ( Dermacentoros rickettsii)

**Name**

- Exanthematic Typhus
- Murine Typhus
- Tsutsugamushi Disease (Scrub Typhus)
- Rocky Mountain Spotted Fever
- Heilig and Naidu's Mysore Typhus
- Kenya Tick Typhus
- South African Tick Fever

**Group Conjectured**

- Weil-Felix OX19 +++
- Weil-Felix OX19 +++
- Weil-Felix OXK +++

<table>
<thead>
<tr>
<th>OX2 ++</th>
<th>OX9 ++</th>
<th>OX8 ++</th>
</tr>
</thead>
<tbody>
<tr>
<td>+ or -</td>
<td>+ or -</td>
<td>+ or -</td>
</tr>
</tbody>
</table>
MAIN GROUPS OF TYPHUS LIKE FEVERS.

CLASSIFICATION OF CASES IN THE INDIAN LITERATURE DIAGNOSED ON CLINICAL GROUNDS.

ONE MAY SHORTLY DISCUSS HERE THE CRITERIA WHICH ONE LAYS DOWN IN AN ATTEMPT TO CLASSIFY THE VARIOUS CASES IN WHICH ANCILLARY INFORMATION IS LACKING. CLINICAL DESCRIPTIONS, HOWEVER ACCURATE, DO NOT GIVE MANY OUTSTANDING POINTS ON WHICH A DIAGNOSIS MAY BE BASED. HOWEVER THE FOLLOWING POINTS MAY BE CONSIDERED TO BE COMPATABLE WITH TYPHUS, PARTICULARLY IF A NUMBER OF CASES ARE SEEN AT ONE TIME.

CASES HAVE BEEN SHOWN TO BE RELATED TO ROCKY MOUNTAIN SPOTTED FEVER BY COMPLIMENT FIXATION TESTS. GENERAL GLANDULAR ENLARGEMENT WAS A FREQUENT FINDING IN REPORTS OF EPIDEMICS OF TSUTSUGAMUSHI DISEASE IN ASSAM AND BURMA BY TATTERSALL (308), GURBUKSH SINGH (109) AND KLEIN (138). AN ESCHAR IS SUGGESTIVE OF A SCRUB TYPHUS INFECTION. THE 'TACHE NOIR' OF FIEVRE BOUTONNEUSE IS VERY SIMILAR. AN ESCHAR IS NOT INVARIABLY SEEN IN SCRUB TYPHUS. MOST CASES OF SCRUB TYPHUS HAVE AT ANY RATE FLEETING LUNG SIGNS, IN TATTERSALL'S (308) SERIES 68%. THE LOCUS OF POSSIBLE INFECTION MAY BE SUGGESTIVE. A JUNGLE INFECTION IS CONSISTENT WITH SCRUB TYPHUS. AN INFECTION IN AN URBAN AREA, IN ASSOCIATION WITH RATS, IS CONSISTENT WITH MURINE TYPHUS. AN INFECTION CONTRACTED IN THE HILLS IS CONSISTENT WITH LOUSE-BORNE TYPHUS. THE SEASON OF THE YEAR MAY BE SUGGESTIVE IN AN EPIDEMIC. SCRUB TYPHUS WAS A DISEASE OF THE MONSOON AND POST-MONSOON IN THE BRITISH ARMY IN BURMA ACCORDING TO SAYERS (276), THOUGH MACKIE (169) REPORTS THAT THE AMERICANS IN BURMA ENCOUNTERED CASES ALL THE YEAR ROUND. COVELL (52) HAS NOTED THAT OXK TYPHUS IN THE SIMLA HILLS, APPEARED FOR TWO MONTHS AT THE END OF THE MONSOON. LOUSE-BORNE AND FLEA-BORNE TYPHUS MAY BE EXPECTED TO BE MORE COMMON IN THE COLDER PARTS OF THE YEAR. THE
### TABLE ELEVEN

**SUMMARY OF THE CLINICAL FINDINGS IN THE EARLY CASES IN THE INDIAN LITERATURE WHICH SUGGEST TYPHUS.**

<table>
<thead>
<tr>
<th>Type of Fever</th>
<th>Continued</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration</td>
<td>About two weeks</td>
</tr>
<tr>
<td>Toxaemia</td>
<td>Marked</td>
</tr>
<tr>
<td>Other Relevant Investigations</td>
<td>Negative</td>
</tr>
<tr>
<td>Rash</td>
<td>Distinctive</td>
</tr>
</tbody>
</table>

**SUMMARY OF POINTS WHICH SUGGEST TO WHICH GROUP OF TYPHUS LIKE FEVERS ANY SERIES OF CASES MAY BELONG**

1. Centrifugal Onset of Rash → Centripetal
   - Rocky Mountain Spotted Fever Group
   - Scrub Typhus
   - Classical Typhus
   - Murine Typhus

2. General Adenitis
   - Scrub Typhus

3. Bronchitis or Pneumonitis
   - Scrub Typhus

4. Locus of Infection
   - Jungle Towns
   - Scrub Typhus
   - Murine Typhus
   - Classical Typhus

5. Season of the Year
   - Monsoon and Post Monsoon
   - Scrub Typhus
   - Classical Typhus
   - Murine Typhus
MORTALITY RATES ARE NOT HELPFUL. PIJPER AND DAU (247), AS MENTIONED PREVIOUSLY, HAVE POINTED OUT IN THE CASE OF SOUTH AFRICAN LOUSE-BORNE AND FLEA-BORNE TYPHUS, THAT THESE DISEASES DO NOT EXACTLY RESEMBLE THEIR COUNTERPARTS ELSEWHERE. IT WOULD NOT BE SURPRISING IF TYPHUS LIKE FEVERS IN INDIA SHOWED VARIATIONS ALSO, AND THE SUGGESTIONS MADE FOR THE CLASSIFICATION OF CASES IN THE LITERATURE, ARE PURELY TENTATIVE. THE SUGGESTIONS ARE SUMMARIZED IN

TABLE ELEVEN.

1. THE INDIAN LITERATURE BEFORE THE STANDARDIZATION OF THE WEIL-FELIX REACTION.

HIRSCH (122) IN 1881 STATED THAT INDIA, FURTHER INDIA AND THE EAST INDIAN ARCHIPELAGO APPEARED TO BE FREE FROM TYPHUS.

CHEVERS (48) IN 1879 SAID THAT HE HAD NEVER SEEN TRUE TYPHUS IN SOUTH BENGAL, THOUGH HE HAD SEEN TWELVE CASES VERY LIKE TYPHUS, DIAGNOSED BY AN INDIAN COLLEAGUE IN CALCUTTA. ELEVEN OF THE TWELVE CASES HAD A MULBERRY LIKE RASH. IT IS CERTAIN THAT ONLY CASES WHICH RESEMBLED THOSE SEEN IN EUROPEAN EPIDEMICS WOULD BE DIAGNOSED AS TYPHUS.

HIRSCH (122) IN HIS CHAPTER ON RELAPSING FEVER AND BILIOUS TYPHOID, REFERS TO MANY EPIDEMICS OF RELAPSING FEVER IN JAILS AND ASYLUMS. HE REFERS TO WALKER'S (324) DESCRIPTION OF
typhus in the north west provinces of india, based on observations on cases seen in the central prison atagra, and considers that what walker saw was relapsing fever. walker's description is of an extremely virulent contagious fever of sudden onset. it seems likely that it was spread by lice or fleas. in view of the definite relapses, and absence of a rash in any case, louse-borne relapsing fever would seem to be a more probable diagnosis than typhus. this was the opinion of boyes-smith, (27) the reviewer of walker's article, referred to by chevers (48).

surveys of the early literature in india have been made by husband and mcwatters (125) in 1908, and covell (52) in 1936. they refer to epidemics reported at the indian congress of medical science in 1852 and 1853.

farquhar, (82) quoted by husband and mcwatters (125) reported an outbreak of typhus in the yusufzie valley, 40 miles north of peshawar. "it succeeded an unusual amount of intermittent or remittent fever". jaundice was noted in most cases from the start as a first and principal feature of the disease. relapses were frequent, with great prostration. the exhibition of quinine was considered to be effective in relapses, not otherwise. (incidentally one may
MENTION IN PASSING THAT THIS STATEMENT DOES NOT SUGGEST THAT THE FEVER WAS NECESSARILY MALARIA.) GREAT EMACIATION WAS ALSO NOTED.

LYELL (164) SEEMED TO THINK THAT THIS FEVER DESCRIBED BY FARQUHAR WAS VERY LIKE THE EDINBURGH RELAPSING FEVER OF 1843-44. IT IS OF COURSE WELL KNOWN THAT LOUSE-BORNE RELAPSING IS A NOT INFREQUENT CONCOMITANT OF LOUSE-BORNE TYPHUS FEVER.

HUSBAND AND MOWATTERS (125) REFER TO REPORTS BY FAIRWEATHER (81) OF TYPHUS IN RAWAL-PINDI JAIL IN 1869, AND TO THE EPIDEMICS REPORTED AT THE INDIAN MEDICAL CONGRESS IN 1894. AT THAT CONGRESS HENDLY AND PISANI STATED THAT TYPHUS WAS ENDEMIC IN THE TRANS-INDUS DISTRICTS, FROM BALUCHISTAN TO YUSUFZAI, HAZARA AND THE HIMALAYAN HILL TRACTS. THESE EPIDEMICS WOULD BE CONSIDERED TO BE TYPHUS BECAUSE OF THEIR RESEMBLANCE TO CLASSICAL EUROPEAN TYPHUS, THE CLINICAL PICTURE OF WHICH WAS THEN WELL DESCRIBED AND WELL KNOWN.

CRAWG (60) IN 1922 SAID THAT A SANITARY OFFICER IN THE KUAMON HILLS, WHO WAS FAMILIAR WITH EUROPEAN TYPHUS, HAD NO DIFFICULTY IN MAKING A DIAGNOSIS WHEN HE SAW CASES THERE. WITH THE EXCEPTION OF THOSE SEEN BY WALKER AND CHEVERS, ALL THESE CASES WERE REPORTED FROM THE FRONTIERS OF INDIA.

SEE MAP ONE.
MAP ONE.
A MAP SHOWING THE AREAS WHERE THE EARLIEST CASES
CONSIDERED TO BE TYPHUS WERE SEEN, WITH A
SUGGESTION AS TO THE POSSIBLE VECTOR.

[Map of South Asia showing various areas and pathways]

? LOUSE... IN THE TRANS INDUS REGION, HENDLY AND PISANI 1894.
QUOTED BY HUSBAND AND MEWATERS 1905.

? FLEA... UNKNOWN VECTOR
HUSBAND AND MEWATERS 1905.

? LOUSE, FAIRWEATHER 1869
QUOTED BY
HUSBAND AND MEWATERS 1905.

? LOUSE, CHEVERS 1879

? LOUSE, THOMPSON 1937.
THEY WERE SEEN AMONG HILL PEOPLES, MUCH
MORE FREQUENTLY LOUSY, AND NOT SO WELL ACQUAIN-
TED WITH SOAP AND WATER AS THE NATIVES OF THE
PLAINs. TYPHUS IS STILL REPORTED FROM THE
FRONTIERS BY THE DIRECTORS OF PUBLIC HEALTH,
INDIA, AS QUOTED BY CHALGRÉN AND BAKER (47).
IN VIEW OF THE FACT THAT TYPHUS IS TO THIS DAY
RECOGNISED IN THESE AREAS, ADMITTEDLY USUALLY
DIAGNOSED ON CLINICAL GROUNDS, AND AS THE
OLDER DESCRIPTIONS WERE BASED ON KNOWLEDGE OF
THE WELL KNOWN EXANTHEMATIC TYPHUS OF EUROPE,
AND AS THE OUTBREAKS WERE SEEN IN A POPULATION
KNOWN TO HARBOUR LICE, I CONSIDER THAT ONE MAY
REASONABLY REGARD THESE EARLY EPIDEMICS AS
LOUSE-BORNE TYPHUS. IT IS POSSIBLE THAT IN
SOME CASES LOUSE-BORNE RELAPSING FEVER WAS
ALSO PRESENT. WALKER'S (324) CASES AND
FARQUHAR'S (82) CASES QUOTED BY HUSBAND AND
MCWATTERS WOULD SEEM TO BE MORE PROBABLY RE-
LAPSING FEVER, AND NOT TYPHUS. IT IS OF
COURSE POSSIBLE FOR TYPHUS TO APPEAR IN AN
EPIDEMIC OF RELAPSING FEVER. YACOB (349)
REFERS TO UNPUBLISHED WORK OF LAL AND JACOB IN
1923, WHO NOTED THAT IN THE COURSE OF THEIR
WORK DURING AN EPIDEMIC OF RELAPSING FEVER
WHICH RAVAGED THE RURAL AREAS OF MOZEFFAGARAH
DISTRICT ON THE EAST BANK OF THE INDUS, A
CERTAIN NUMBER OF CASES IN A PARTICULAR AREA
SUfFered FROM A CONTINUED FEVER WITH A POSITIVE
WEIL-FELIX REACTION. THERE IS ALSO A POSSIBILITY HOWEVER THAT THESE WERE CASES OF RELAPSING FEVER WITH A POSITIVE WEIL-FELIX AS REPORTED BY ELSDON-DEW (77).

SEE MAP TWO.

HUSBAND AND MCMATTERS (125) IN 1908 CONSIDERED THAT THE TRANS-INDUS REGION WAS LIABLE TO TYPHUS EPIDEMICS FROM TIME TO TIME, AND IT WAS PROBABLY "ENDEMIC" THERE.

SEE MAP ONE.

THEY PUBLISHED IN 1908 A DESCRIPTION OF ONE HUNDRED AND TWENTY-FOUR CASES. THESE CASES OCCURRED DURING THE WINTER MONTHS IN 1905, MAINLY AMONGST THE PERSONNEL OF TWO MULE CORPS IN PESHAWAR. THE CASES WERE CHARACTERISTICALLY APATHEtic WITH CONGESTED EYES AND IN 80% OF CASES HAD A FAINT BODY RASH. THE MORTALITY IS NOT MENTIONED, HENCE ONE PRESUMES THAT IT WAS NOT HIGH. ACCORDING TO FREIDMAN (99) THE MORTALITY OF LOUSE-BORNE TYPHUS MAY VARY, BUT IT IS GENERALLY CONSIDERED THAT IN EPIDEMICS THE MORTALITY IS HIGH. NO LICE WERE FOUND AND ONE HUNDRED AND TWENTY OF THE CASES WERE SOLDIERS WHOSE CLEANLINESS AND HABITS IN PEACE TIME SHOULD EXCLUDE LICE. ONE CANNOT, I THINK, EXCLUDE THE RAT FLEA AS A POSSIBLE VECTOR, AND IT DOES NOT SEEM IMPOSSIBLE FOR THE FIRST MULE CORPS COMING FROM TIBET IN 1905, TO HAVE BROUGHT INFECTED RATS OR FLEAS IN THEIR BAGGAGE. IT IS
MAP TWO

LOCATION IN INDIA OF MAP SHOWN BELOW.

AREA WHERE IN 1923 LAL AND YACOB OBSERVED CASES OF CONTINUED FEVER WITH A POSITIVE WEIL-FELIX REACTION DURING AN EPIDEMIC OF RELAPSING FEVER. THE DIAGNOSIS IS UNCERTAIN AS IT IS NOW KNOWN THAT A POSITIVE WEIL-FELIX REACTION IS FOUND IN CASES OF RELAPSING FEVER.
TRUE THAT WE HAVE TO THIS DAY NO CERTAIN KNOWLEDGE THAT MURINE TYPHUS IS PRESENT IN TIBET.

HUSBAND AND MCGWATTERS REFER TO EPIDEMIC PNEUMONIA REPORTED FROM MALAKAND AND OCCURRING IN A WAZIRISTAN EXPEDITION IN 1895. IN MALAKAND THERE WAS A "PNEUMONIA" BARRACKS. IN THE WAZIRISTAN EXPEDITION THE MAJORITY OF THE FIFTY CASES DIED, INCLUDING ELEVEN OUT OF THIRTEEN OF THE SICK ATTENDANTS. THIS DESCRIPTION, IF IT WERE NOT FOR THE ABSENCE OF A RASH, SEEMS CONSISTENT WITH EXANTHEMATIC TYPHUS FEVER. HUSBAND AND MCGWATTERS' REPORT SUGGESTS THAT SOME OF THE CASES AND EPIDEMICS OF TYPHUS-LIKE FEVER EVEN IF SPREAD BY THE LOUSE MAY NOT BE EXACTLY SIMILAR TO EUROPEAN LOUSE TYPHUS, IN THE SAME WAY AS PIJPER AND DAU (247) CONSIDER THAT SOUTH AFRICAN LOUSE TYPHUS IS NOT EXACTLY SIMILAR TO THE EUROPEAN VARIETY.

HEPPER (121) REPORTED A SMALL OUTBREAK IN 1905 IN THE JAIL IN PESHAWAR. ALL FIVE CASES HAD BEEN IN JAIL FOR TWO MONTHS AND PRESUMABLY INFECTION MUST HAVE BEEN CONTRACTED THERE. FOUR OF THE FIVE HAD BEEN LIVING IN THE JAIL HOSPITAL. THREE ATTENDANTS, WHO WOULD PRESUMABLY HAVE HAD OUTSIDE CONTACTS, HAD BEEN SICK HOWEVER. ONE IS INCLINED TO SEEK FOR A CONNECTION BETWEEN THESE CASES AND THOSE REPORTED BY HUSBAND AND MCGWATTERS. ALL THE CASES HAD AN EXTENSIVE RASH IN CONTRAST TO THE FAINT RASH.
SEEN IN HUSBAND AND MOWATTERS' CASES, AND ONE DIED. A SEARCH FOR THE VECTOR REVEALED BED BUGS IN THE IRON COTS. AFTER AN INTENSIVE ATTACK ON THE BUGS AND ISOLATION OF THE PATIENTS, ONLY ONE MORE CASE OCCURRED SIX DAYS LATER. THE INCUBATION PERIOD OF THIS LAST CASE MIGHT WELL HAVE BEEN MORE THAN SIX DAYS. THE ASSOCIATION WITH THE HOSPITAL SUGGESTS A VECTOR THERE, WHICH WAS DISPOSED OF WHEN THE ATTACK WAS MADE ON THE BED BUGS. ONE MAY LEGITIMATELY SUSPECT RAT FLEAS. AS HEPPER WAS AWARE, BED BUGS HAVE NEVER BEEN IMPLICATED IN THE TRANSMISSION OF TYPHUS, AND HE SUGGESTED THAT FURTHER INVESTIGATION WAS NECESSARY.

BASU (17) IN 1927 REPORTED CASES DESCRIBED BY MITRA IN 1912 AND 1917 IN CALCUTTA AND CONSIDERED TO BE "PSEUDO-TYPHUS".

IN 1912 BRADLEY AND SMITH (29) REPORTED A CURIOUS CASE OF FEVER OCCURRING IN A BRITISH SOLDIER STATIONED NEAR BARRAKPUR ABOUT TWELVE MILES FROM THE CENTRE OF CALCUTTA. THEY CONSIDERED THAT IT WAS POSSIBLY A CASE OF TYPHUS. THEIR LABORATORY FINDINGS WERE NEGATIVE. THEY RECORDED IT AS A CASE OF PYREXIA OF UNKNOWN ORIGIN. THERE SEEMS EVERY LIKELIHOOD THAT IT WAS A CASE OF FEVER OF THE TYPHUS GROUP, VECTOR UNCONJECTURED AND GROUP UNKNOWN.

MEGAW (186) REPORTED HIS OWN ATTACK OF FEVER IN 1917, WHICH HE HAD CONTRACTED IN THE
KUAMON HILLS IN THE PREVIOUS YEAR. HE CONSIDERED THAT THE FEVER RESEMBLED BRILL’S DISEASE, AND THAT IT ALSO WAS LIKE THE MILD IDAHO TYPE OF ROCKY MOUNTAIN SPOTTED FEVER. HE REFERRED TO McKECHNIE’S (181) CASES SEEN IN THE SAME AREA. McKECHNIE HAD SEEN HIS CASES IN BHIM TAL AND SAT TAL, NEAR NAINI TAL. THESE STATIONS GEOGRAPHICALLY RESEMBLE PESHAWAR BEING SITUATED AT THE ENTRANCE TO A MAIN PASS LEADING TO THE NORTH. BLEWITT (26) HAS STRESSED THE NOMADIC LIFE OF THE NATIVES AND THE MOVEMENT OF PILGRIMS THROUGH THE AREA, AND HAS SAID THAT THERE IS A CONSIDERABLE AMOUNT OF TYPHUS ENDEMIC AMONG THE HILL PEOPLE, WHOSE HABITS FAVOUR THE SPREAD OF DISEASE BY LICE OR FLEAS OR BOTH. McKECHNIE’S CASES, AS DESCRIBED IN MEGAW’S (187) LATER ARTICLE, ARE WITHOUT A DOUBT CASES OF TYPHUS LIKE FEVER. CLINICALLY THEY RESEMBLE CASES DESCRIBED LATER BY MANY OBSERVERS IN INDIA. MEGAW (187) SAID “THE IMPORTANCE OF McKECHNIE’S REPORT IS THAT IT CONTAINS THE ONLY RECORD KNOWN TO ME OF A NUMBER OF CASES OF WHAT I THINK TO BE A PREVIOUSLY UNRECOGNISED DISEASE IN INDIA. FOR THOUGH I ENTIRELY AGREE WITH McKECHNIE THAT HIS CASES WERE CLINICALLY INDISTINGUISHABLE FROM TYPHUS, A DISEASE WHICH HAD BEEN OBSERVED BY A NUMBER OF MEDICAL MEN IN INDIA, I THINK THERE IS REASON TO BELIEVE THAT THESE CASES SHOULD BE
CLASSIFIED WITH THE ROCKY MOUNTAIN SPOTTED FEVER, A DISEASE WHICH DIFFERS VERY WIDELY FROM TYPHUS IN ITS EPIDEMIOLOGY THOUGH ITS SYMPTOMS VERY CLOSELY RESEMBLE THOSE OF TYPHUS”.

KEATES (135) IN 1920 SAW A CASE WHICH HE CONSIDERED TO BE TYPHUS IN MUREE, A HILL STATION IN THE EXTREME NORTH OF THE PUNJAB. THIS CASE IS OF INTEREST AS THE RASH APPEARED CENTRIFUGALLY, AND WAS NOT PROMINENT ON THE TRUNK, THE ABDOMEN AND CHEST REMAINED FREE FROM THE RASH. THIS RESEMBLES THE RASH OF THE TYPHUS-LIKE FEVERS LATER DESCRIBED BY HEILIG AND NAIDU (117 & 118) IN MYSORE IN SOUTH INDIA. THIS CASE MIGHT BE PROVISIONALLY GROUPED IN THE INDETERMINATE GROUP, BECAUSE OF THE CENTRIFUGAL DEVELOPMENT OF THE RASH. MEGAW (137) IN 1921 REPORTED FIVE FURTHER CASES WHICH HAD BEEN REPORTED TO HIM. HE DISCUSSED THESE CASES, HIS OWN ILLNESS REPORTED IN 1917, AND McKECHNIE’S CASES. THREE CASES WERE SEEN IN NAGPUR BY LT-COL.CHAPMAN; ONE CASE WAS SEEN IN CAWNPORE BY COL.HARDY; ONE CASE WAS SEEN IN BANGLAORE BY LT-COL.SPRAWSON; AND ONE CASE WAS SEEN IN BANGLAORE BY MAJOR BROADRIBB. ALL THESE LATTER CASES WERE CONSIDERED BY MEGAW TO RESEMBLE HIS OWN ILLNESS AND McKECHNIE’S CASES. MEGAW MENTIONED THE VARIATIONS IN SEVERITY OF CASES OF ROCKY MOUNTAIN SPOTTED FEVER AND SPECULATED WHETHER THE SEvere TYPE ORIGINATED FROM CLASSICAL
Typhus, and the milder variety from the milder disease of Brill.

In a note on the twelve day fever of Nigeria Megaw (188) in 1921 pointed out the clinical resemblance of the Indian cases reported by him, to those cases in Nigeria reported by Davies and Johnson (64) and the earlier cases of "anomalous" paratyphoid fevers in South Africa reported by McNaught (183).

Cragg (60) in 1922 discussed Megaw's prima facie case against the tick, and preferred to make a prima facie case against the louse, while not disputing the possibility of the tick being the vector.

Jackson (127) in 1924 reported five cases of typhus fever observed since 1915, from the hills in the south of the Bombay Presidency. The vector was not discovered, and no opinion can be given on the possible group to which they might belong.

Cunningham and Theodore (62) reported in 1924 eight cases of typhus fever seen in the Madras Presidency amongst a detachment of the Assam Rifles, recently arrived from Calcutta. They considered that the disease might have been acquired in that city. Megaw and Sunder Rao (192) considered this unlikely and one is inclined to agree with them. It seems much more likely that
THIS TYPHUS WAS ACQUIRED IN THE JUNGLE, AND MAY HAVE BEEN TYPHUS OF THE INDETERMINATED GROUP OF UNKNOWN VECTOR, THOUGH MITE-BORNE SCRUB TYPHUS IS NOT IMPOSSIBLE; THE VERY DISTINCT RASH IN DARK SKINS HOWEVER IS UNLIKE THE USUAL SCRUB TYPHUS ERRUPTION.

BASU (18) REPORTED FIFTEEN CASES OF TYPHUS FROM CALCUTTA IN 1924. THESE CASES WERE SEEN IN THE SLUM AREAS NEAR THE HOOGLI RIVER AND THE APPEARANCE OF SEVERAL CASES IN A HOUSE SUGGESTED A LOUSE OR FLEA VECTOR. THE MORTALITY WAS NIL. THE CASES WERE SEEN IN THE CALCUTTA COLD SEASON BETWEEN NOVEMBER AND JANUARY. THOUGH BASU SEEMED TO BE CONVINCED THAT IT WAS LOUSE-BORNE TYPHUS, AND INDEED LICE WERE FOUND ON THREE CASES, I WOULD THINK THAT IT WAS MORE LIKELY TO BE MURINE TYPHUS.

PHIPSON (245) IN 1924 REPORTED AN OUTBREAK IN SIMLA. SIXTEEN CASES WERE SEEN, ALL IN ONE FAMILY. THE SOURCE OF INFECTION WAS THE NEIGHBOURING STATE OF BHAIJI, WHERE A LIMITED OUTBREAK OF WHAT WAS BELIEVED TO BE TYPHUS WAS IN PROGRESS. THE CLINICAL DESCRIPTION IS SUGGESTIVE ENOUGH, THOUGH CONFIRMATORY WEIL-FELIX TESTS WERE POSITIVE IN ONLY SOME OF THE CASES TO HIGH TITRE. THE DETAILS GIVEN OF THE TECHNIQUE OF THE TEST BY PHIPSON (246) SUGGESTS THAT SOME OF THE NEGATIVE RESULTS MAY
HAVE BEEN DUE TO THE METHOD EMPLOYED. THE LOCAL HILL MEN WERE SAID TO RECOGNISE A FEVER PREVALENT IN THOSE PARTS WHICH SPREADS IN AND IS LOCALIZED TO A FAMILY. IN THIS PARTICULAR FAMILY, ALL OF WHOM WERE VERMINOUS, THREE MEMBERS DID NOT CONTRACT THE DISEASE. IT IS INTERESTING TO NOTE THAT THIS IS THE FIRST DESCRIPTION OF A TYPHUS-LIKE FEVER IN INDIA BEING DIAGNOSED IN THE ABSENCE OF A RASH. A RASH WAS ONLY OBVIOUS IN TWO CASES, THOUGH IT COULD BE MADE OUT IN FOUR OTHER CASES. SIX OF THE SIXTEEN CASES PERISHED. THE LOCALITY OF THE OUTBREAK, THE CLINICAL DESCRIPTION, AND THE APPARENT SPREAD IN A VERMINOUS FAMILY SUGGEST EXANTHEMATIC TYPHUS, WHICH IS CONFIRMED BY THE FAIRLY HIGH MORTALITY. THE WEIL-FELIX RESULT IS CONSISTENT WITH THAT OPINION. MEGAW (189) IN 1924 AGAIN DISCUSSED THE NOMENCLATURE OF TYPHUS LIKE FEVERS. HE CONSIDERED THE "PLACE" NAMES UNSATISFACTORY. HE THOUGHT THAT THE PSEUDO-TYPHUS OF SUMATRA, THE MOSSMAN FEVER OF AUSTRALIA REPORTED BY SMITHSON (289) AND THE ADELAIDE FEVER OF HONE (123 & 124) MIGHT BE MITE TYPHUS. IT WAS TO BE SHOWN LATER THAT THE TWO FORMER WERE OF THAT GROUP AND THE LATTER WAS MURINE TYPHUS. HE REFERRED TO CRAGGS (60) CRITICISMS BUT WISHED TO MAKE HIS A "PRIMA FACIE" CASE AGAINST THE TICK. HE MENTIONS THAT MCNAUGHTS, (183) AND DAVIES
AND JOHNSON'S (64) PATIENTS WERE BITTEN BY TICKS, AND THEREFORE THE TICK WAS POSSIBLY THE VECTOR. THIS FACT WAS OF COURSE OF INTEREST. IN THE CASES REPORTED IN 1921, TWO CASES SEEN BY CHAPMAN HAD BEEN LIVING IN PLAGUE HUTS (ISOLATION HUTS) IN CLOSE PROXIMITY TO JUNGLE. THE RASH IN CHAPMAN'S THIRD CASE WAS VERY DISTINCT ON THE LEGS AND BECAME PETECHIAL. IT WOULD SEEM POSSIBLE THAT IT MAY HAVE HAD A CENTRIFUGAL ONSET. IN SPRAWSON'S CASE A TICK WAS SEEN. IN JULY 1923 MEGAW FOUND A TICK ON HIS LEG WHILE IN BHIM TAL, IDENTIFIED BY STRICKLAND AS RHIPICEPHALUS SANGUINEUS. HE MENTIONS THAT McKECHNIE HAD SAID THAT "THE LIABILITY TO CONTRACT INFECTION APPEARS TO HAVE BEEN DIRECTLY IN PROPORTION TO THE DEGREE IN WHICH THE RESIDENTS WERE COMPelled TO LIVE IN ASSOCIATION WITH THE LIFE OF THE JUNGLE", THERE WAS ONE EXCEPTION TO THE RULE THAT THE CASES WERE ISOLATED. FIVE SERVANTS AND HIS OWN DAUGHTER CONTRACTED THE DISEASE IN McKECHNIE'S HOUSE. MEGAW MENTIONED MITRA'S (202) REPORT OF CASES IN HOWRAH IN 1912. WILLIAMSON (336) IN 1925 REPORTED A CASE FROM SAUGOR. THE RASH WAS PROMINENT ON THE PALMS AND SOLES AND WAS PETECHIAL ON THE LEGS. IT WAS ALSO PRESENT ON THE TRUNK. THE CAMP WHERE THE DISEASE WAS PROBABLY CONTRACTED, WAS SAID TO HAVE BEEN "FULL OF HARD TICKS".
This case may be classified as one of typhus like fever, group indeterminate, with suspicion directed to the tick as a vector. The case is referred to by Megaw, Shettle and Roy (190).

Norman and Ramachandran (229) reported a case from Trichinopoly in 1925. The rash which began on the legs almost immediately spread to the trunk. It seems likely that the patient contracted the disease in Trichinopoly. The type of the disease and the possible vector remain in doubt, though the centrifugal onset of the rash may be significant, and provisionally it may be classified in the group indeterminate. This case is also referred to by Megaw and Rao (192).

Megaw, Shettle and Roy (190) in 1925 reported sixteen cases seen in Saugor in central India. All the patients were soldiers, fourteen were Europeans, two Indians. They contracted the disease while on manoeuvres. There was no history of local disease, and the chance distribution, absence of a history of a bite, or a sign of a vector, suggests that this infection might have been the typhus of indeterminate group. The authors considered that the absence of a sore, absence of local lymphadenitis and absence of evidence of the existence of biting mites, excluded mite typhus. Later reports have shown of course that these
CLINICAL FEATURES ARE SOMETIMES ABSENT, BUT NOT AS A RULE IN EUROPEANS. THE NEGATIVE RESULTS OF GUINEA PIG INOCULATION IS CONSISTENT WITH EITHER INFECTION. THE DISTRIBUTION OF THE RASH, PROMINENT ON THE WRIST AND PALMS AND SOLES IS CONSISTENT WITH THE TYPHUS OF UNKNOWN VECTOR LATER SEEN AND DESCRIBED BY HEILIG AND NAIDU (117, 118 & 119) IN MYSORE.

MEGAW (191) IN 1925 REPORTED SEVEN FURTHER CASES WITH NEGATIVE ANIMAL INOCULATION RESULTS. THESE CASES HAD BEEN SEEN BY VARIOUS OBSERVERS IN ORISSA, (MUERJI) DACCA, (ANDERSON) AKYAB IN BURMA, (WATERS) SAUGOR (WILLIAMSON'S CASE) AND BALAGHAT (BRANDON AND BOYD AND BARNARDO AND FRANCAIS) IN THE CENTRAL PROVINCES. IN MOST OF THE CASES THE TICK COULD HAVE BEEN THE VECTOR. WHILE VISITING ONE OF THESE AREAS, MEGAW NOTICED AND SECURED, A TICK ON HIS LEG, WHICH WAS IDENTIFIED AS RICICEPHALUS SANGUINEUS, WHICH IS THE VECTOR OF FIEVRE BOUTONNEUSE. IT RESEMBLED THE TICK WHICH HAD BITTEN HIM THREE WEEKS BEFORE HIS OWN ATTACK OF TYPHUS IN THE KUMAON HILLS.

MCWATTERS (185) MISTAKENLY PRINTED AS BANNERJEE, IN 1927 REPORTED TWO CASES FROM THE KUMAON, ONE OF WHICH WAS FATAL. THESE TWO CASES COULD BE SAID TO RESEMBLE THE OTHER CASES OF UNKNOWN VECTOR FROM THE KUMAON REPORTED BY McKECHNIE (181)
Mackenzie (168) in 1927 described two cases occurring in the North West Frontier Province, which though not investigated were suggestive of typhus. The rash appeared centrifugally. Though the district is where louse-borne typhus is seen, the patients, a British officer and his child were unlikely to be in contact with infected lice, much less to harbour them. Mackenzie records the description given to him of an epidemic of fifteen cases with eight deaths, of what seemed a similar disease, which originated in a house to which one of the servants of his patients had returned a week before the epidemic had started. The centrifugal origin of the rash suggests the indeterminate group.

Bannerji (14) reported in 1927, a case suggestive of typhus fever from Allahabad. There was a rash on the sixth day beginning on the chest and becoming generalised. There was congestion of the lungs. Recovery was rapid.

Linderman (160) reported two cases in 1928, from Hyderabad Sind and Quetta. Scrub typhus was possible, but the description of the rash resembled the rash of a case seen by myself in Calcutta which was serologically OX2. I would regard these as cases of the indeterminate group of unknown vector.
IN 1928 MEGAW AND SUNDER RAO (192) DISCUSSED THE NOMENCLATURE AND THE VECTOR PROBLEM IN AN ARTICLE ON THE SPORADIC FEVERS OF THE TYPHUS GROUP. THEY DISCUSSED CASES REPORTED IN INDIA, AS WELL AS PERSONAL REPORTS FROM VARIOUS OBSERVERS IN INDIA. THEY REFERRED TO THE WORK OF FLETCHER IN MALAYA USING THE KINGSBURY ('K') STRAINS. THEY MENTION THE CASES DESCRIBED BY ANDERSON (6) IN KENYA; THE SO CALLED CASES OF "BRILLS DISEASE" DESCRIBED IN MARSEILLES BY BURNET AND OLMER (40); AND MAXCY'S (177 & 178) ENDEMIC TYPHUS IN ALABAMA. SHORT NOTES ON CASES SEEN BY VARIOUS OBSERVERS IN INDIA WERE GIVEN. SOME OF THESE CASES HAD BEEN BITTEN BY TIKKS. THESE CASES WERE SEEN IN RUTLAM IN THE CENTRAL PROVINCES (SCOTT), SECUNDERBAD (FIELDING), BANGALORE (MACPHERSON, AND TWO OTHERS IN PESHWAR AND BAGDAD), MUTKULI, IN THE CENTRAL PROVINCES (HAMILTON), SAUGOR (DOTIVALA), SAUGOR (SHETTIE), SAUGOR (ROY AND RAO) SAUGOR (RAO), DARJEELING (WALKER) BALAGHAT IN THE CENTRAL PROVINCES (DOYLE), ALLAHABAD (GHOSH) BHIM TAL (RAO)

BHIM TAL (THAPPA). OTHERS MENTIONED WERE REPORTED INDEPENDENTLY.

MEGAW AND RAO THEN DREW ATTENTION TO THE VARIOUS PARTS OF THE WORLD WHERE TYPHUS LIKE FEVERS FOLLOWED THE BITES OF TIKKS, BEGINNING WITH ROCKY MOUNTAIN SPOTTED FEVER
WHERE IT WAS THE PROVED VECTOR. THE LOW MORTALITY, THE NEGATIVE REACTIONS WITH GUINEA PIGS AND THE TOTAL ABSENCE OF EVIDENCE TO SUGGEST PERSON TO PERSON INFECTION WAS CONTRASTED WITH THE FINDINGS IN LOUSE BORNE TYPHUS. THEY NOTED OF COURSE THAT THE ACTUAL EVIDENCE TO SUGGEST AN ARTHROPOD VECTOR WAS SURPRISINGLY SCANTY. IT WAS EVIDENT TO THEM THAT THE WEIL-FELIX RESULTS WERE CONFLICTING. FOR THE HYPOTHESIS THAT THE TICK WAS THE VECTOR IN INDIA MEGAW AND RAO POINTED OUT THAT TICKS COULD POSSIBLY BE THE VECTOR AS THE BITE WAS USUALLY PAINLESS, AND TICKS WERE NOT SUSPECTED. MAN WAS NOT THEIR NATURAL HOST. THEY THOUGHT THE MITE AN UNLIKELY VECTOR IN VIEW OF THE ABSENCE OF A LOCAL SORE AND LOCAL ADENITIS, WHICH AT THAT TIME WAS CONSIDERED TO BE ALMOST INVARIABLY PRESENT. THEY EMPHASIZED THE RELATIONSHIP BETWEEN TRUE TYPHUS AND THE TYPHUS LIKE FEVERS, DEMONSTRATED PATHOLOGICALLY BY WOOLBACH IN THE CASE OF LOUSE TYPHUS AND ROCKY MOUNTAIN SPOTTED FEVER. THEY DID NOT AGREE WITH GOODALL HOWEVER, (WHO WISHED TO CLASSIFY ALL TYPHUS LIKE FEVERS IN ONE GROUP), POINTING OUT THE USEFUL PURPOSE OF CLASSIFICATION IN EMPHASIZING THE EPIDEMIOLOGICAL DIFFERENCES. MEGAW (189) IN 1924 HAD SPECULATED WHETHER THE VIRUS OF LOUSE TYPHUS AND 'TICK' TYPHUS HAD A COMMON
ORIGIN, AND WONDERED WHETHER EXCEPTIONALLY, EITHER MIGHT BE CONVEYED BY BOTH VECTORS, INSTEAD OF ONE. THUS IN McKECHNIE'S SERIES, THE SIX CASES IN ONE HOUSEHOLD MIGHT HAVE HAD A LOUSE SPREAD INSIDE THE HOUSE. THEY POINTED OUT THAT NICHOLLE AND ANDERSON (222) HAD FOUND THAT THE TICK BORNE SPANISH RELAPSING FEVER COULD BE CONVEYED BY THE LOUSE. THIS ARTICLE SHOWED THAT SPORADIC CASES OF TYPHUS LIKE FEVERS AND OCCASIONAL SMALL LOCAL OUTBREAKS WERE BEING RECOGNISED ALL OVER CENTRAL INDIA, AND IN THE KUMAON. ALL THESE CASES HAD DEFINITE RECOGNISABLE RASHES.


GOSE (104) IN 1928 REPORTED TWO CASES FROM ALLAHABAD IN WHICH THE RASH APPEARED CENTRIFUGALLY. THERE WAS AN INDEFINITE HISTORY OF BITES IN BOTH CASES. THE WEIL-FELIX TEST
WAS NOT CARRIED OUT.

DE (66) IN 1930 REPORTED A SIMILAR CASE, FROM BILASPUR IN WHICH THE WEIL-FELIX TEST WAS NOT CARRIED OUT. THE RASH APPEARED FIRST ON THE FOREARMS AND LEGS. THE CENTRIFUGAL ONSET MAY JUSTIFY THE TENTATIVE PLACING OF THESE CASES IN THE INDETERMINATE GROUP.

LINDBERG (159) IN 1931 REPORTED FIVE CASES SEEN NEAR POONA. ALL WERE MISSIONARIES, ALL WERE COMPLETELY COVERED BY A MACULO-PAPULAR RASH. THE WEIL-FELIX WAS SAID TO BE POSITIVE DURING CONVALESCENCE. THERE WAS NO HISTORY OF A BITE. THE RASH IS RATHER SUGGESTIVE OF SCRUB TYPHUS, BUT I WOULD NOT CONSIDER THAT THE CASES CAN BE PLACED IN ANY GROUP WITH CERTAINTY.

LABARNADIE (143) IN 1932 REPORTED A CASE SEEN DURING A DENGUE EPIDEMIC. HE CONSIDERED THE RASH RESEMBLED THAT OF FIEVRE BOUTONNEUSE. THE WEIL-FELIX WAS IN RAISED TITRE TO SEVERAL PROTEUS SUSPENSIONS INCLUDING AN OXK. INTRAPERITONEAL INOCULATION OF A GUINEA PIG WAS NEGATIVE. IT IS NOT POSSIBLE TO PLACE IT WITH ANY CERTAINTY IN ANY GROUP.

MUKERJI (206) IN 1932 REPORTED THREE CASES WITH RASHES SUGGESTIVE OF TYPHUS IN THE NAGPUR AREA. IT IS DIFFICULT TO COME TO ANY CONCLUSION ON THE SCANTY REPORT.

THOUGH THE KINGSBURY SUSPENSIONS HAD FIRST BEEN USED IN MALAYA IN 1925, IT WAS NOT
Until 1932 that the use of the OxK Proteus was reported from India.

Biggam (21) in 1932 reported three cases from Bangalore. K strains were used in addition to the usual X19 strains. His cases were Britishers and had been in the jungle during the possible incubation period. All three cases had a rash. In two cases the rash began centrifugally in the manner described in Heilig and Naidu's (117 & 118) Mysore cases. The Weil-Felix titres were all low. In one case there was a significant rise of OxK, in another a rise of 1/250 Xk, X 'Warsaw' (X19) and X 'Multesar' (an Indian strain of X19).

Bridges (30) records the origin of X 'Multesar' from Muktesar in the Punjab. The indeterminate serology of low titre and the centrifugal development of the rash are reminiscent of Heilig and Naidu's (117 & 118) cases from Mysore.

In 1932 Kundu (142) reported the first case of typhus-like fever with a firm serological confirmation from Burma. This was serologically positive to Ox19 in 1/2000. Kundu saw a parallel in his case and the descriptions of 'shop' or 'urban' form of tropical typhus reported from Malaya. As cases of the disease had up till then never been recognised in Burma he surmised that the infection had in
SOME WAY BEEN "IMPORTED" FROM MALAYA. THERE
SEEMS VERY LITTLE DOUBT HOWEVER THAT THIS DIS-
EASE WAS PRESENT IN BURMA AND WAS MURINE
TYPHUS, AND THIS WAS THE FIRST REPORTED CASE.

SACHS (270) REPORTED IN 1935 SEVEN
CASES SEEN BETWEEN 1929 AND 1932. ALL WERE
BRITISH AND ALL HAD A GENERALIZED RASH. FIVE
OF THESE CASES WERE SEEN IN JUBBLEPORE. IN
ONLY ONE CASE WAS THE WEIL-FELIX DONE WITH
'O' SUSPENSIONS. THERE WAS A RISE FROM 0 ON
THE SEVENTH DAY OF THE DISEASE TO A TITRE
OF 1/500 ON THE 17TH DAY. THE ONE FATAL CASE WAS
AUTOPSIED. THIS IS ONE OF THE FEW RECORDED
AUTOPSIES OF A TYPHUS-LIKE FEVER IN INDIA
PRIOR TO THE SECOND WORLD WAR. THE MORBID
ANATOMICAL APPEARANCES WERE SOMEWHAT SIMILAR
TO APPEARANCE SEEN BY ME IN CASES OF SCRUB
TYPHUS IN CALCUTTA IN 1943. THE MORBID HIST-
OLOGICAL APPEARANCES WERE OF A VERY ACUTE TOXIC
PROCESS PARTICULARLY IN THE KIDNEYS AND
MYOCARDIUM. IN THE LIVER THERE WAS LOCAL
NECROSIS AND A ROUND CELLED INFILTRATION
MAINLY IN THE PORTAL TRACTS. ARTERIOLITIS AND
PERIARTERIOLITIS WAS SEEN IN THE LIVER, SPLEEN,
AND ENDOCARDIUM. MICROSCOPIC HAEMORRHAGES WERE
NOTED IN THE PANCREAS, THE LIVER, THE SPLEEN,
AND IN THE KIDNEY NEAR THE LOOPS OF HENLE.
THERE WAS A ROUND CELLED INFILTRATION IN THE
PANCREAS AND THE MALPIGHIAN CORPUSCLES OF THE
KIDNEY, THE CELL NUCLEI OF WHICH STAINED FAINTLY. THERE WAS NO INTERSTITIAL NEPHRITIS, INTERSTITIAL MYOCARDITIS NOR PNEUMONITIS DESCRIBED LATER IN SCRUB TYPHUS BY ALLEN AND SPITZ (2) AND OTHERS. IT IS NOT POSSIBLE TO GROUP THESE CASES. AS THEY PROBABLY CONTRACTED THE DISEASE IN THE JUNGLE, SCRUB TYPHUS IS A POSSIBLE DIAGNOSIS, BUT THE WEIL-FELIX RESULT IN THE ONE CASE IN WHICH EFFECTIVE SUSPENSIONS WERE USED DOES NOT SUGGEST THAT THIS IS LIKELY. IT IS UNLIKELY THAT IT WAS EITHER LOUSE-BORNE EXANTHEMATIC OR MURINE TYPHUS. THE MORE LIKELY GROUP WOULD SEEM TO BE THE UNDETERMINED SEROLOGICAL GROUP OF FELIX (86 & 87), THE VECTOR OF WHICH IS UNKNOWN IN INDIA.

IN 1936 SHORTT AND D'SILVA (286) COLLECTED THE REPORTS OF 177 RECORDED CASES OF TYPHUS IN INDIA. IN ONLY SIXTEEN OF THESE WAS THERE A DEFINITE HISTORY OF A TICK BITE. MANIFOLD'S (170) FIGURES QUOTED BY BLEWITT (26) IN 1936 WERE OF 216 CASES IN BRITISH AND INDIAN TROOPS BETWEEN 1933 AND 1935, IN WHICH ONLY TWO GAVE A HISTORY OF TICK BITE. AS ALREADY MENTIONED BLEWITT (26) IN 1936, FAILED TO OBTAIN EVIDENCE OF INFECTION OF TICKS IN THE KUMAON. HEILIG AND NAIDU (117 & 118) IN 1942 FAILED TO FIND EVIDENCE OF RICKETTSIAL INFECTION OF TICKS IN MYSORE. IN MALAYA, LEWTHWAITE, HODGKIN AND SAVOOR (152) WERE UNABLE TO INFECT,
OR TRANSMIT TO THE TICKS DERMACENTOR ANDERSONI OR RHIPCEPHALUS SANGUINEUS, EITHER MALAYAN MURINE (FLEA-BORNE) TYPHUS, OR MALAYAN SCRUB TYPHUS. IT IS TRUE THAT NEITHER OF THESE TWO LATTER FORMS OF TYPHUS HAVE EVER BEEN KNOWN TO BE TRANSMITTED BY A TICK.

IT IS A FACT THAT TICK BITES CAN BE OVERLOOKED EVEN BY NATURALISTS ACCORDING TO SUNDER RAO (306). HE HAS RECORDED CASES OF TICKS BEING ATTACHED UNNOTICED TO INDIVIDUALS FOR DAYS. DYER, CEDER, WORKMAN, RUMREICH AND BADGER (73) HAVE SHOWN THAT GUINEA PIGS CAN BE EXPERIMENTALLY INFECTED WITH MURINE TYPHUS BY RUBBING ABRASIONS WITH INFECTED FLEA TISSUES OR FLEA FAECES.

BANNERJI (15) IN 1937 REPORTED A CASE OF TYPHUS WHICH HE CONSIDERED TO BE "TICK" TYPHUS FROM ALLAHABAD, A RASH WAS SEEN, AND THE CLINICAL DESCRIPTION WAS TYPICAL OF A TYPHUS-LIKE DISEASE. NO WEIL-FELIX TEST WAS CARRIED OUT. IT WAS IMPOSSIBLE TO CONCLUDE WHAT THE POSSIBLE VECTOR MIGHT BE.

THOMPSON (310) IN 1939 REPORTED EIGHT CASES FROM MEERUT IN NORTHERN INDIA. THE FIRST CASE REPORTED SICK FOUR DAYS AFTER HIS RETURN FROM LEAVE TO HIS HOME IN KASHMIR, CLOSE TO ONE OF THE TRADE ROUTES FROM INSIDE ASIA. HE HAD BEEN TO A FESTIVAL FOURTEEN DAYS BEFORE HE DEVELOPED FEVER. TICKS WERE ASSUMED

PRADHAN (256) IN 1944 REPORTED ABOUT FIFTY CASES SEEN IN SIX AND A HALF YEARS IN BERAR, CENTRAL PROVINCES. THE CASES WERE USUALLY SEEN BETWEEN AUGUST AND OCTOBER, WITH A MORTALITY OF ABOUT TEN PER CENT. CLINICALLY THE CASES WERE OF A PATTERN WHICH WOULD SUGGEST SCRUB TYPHUS, WITH A GENERALIZED RASH, TOXAEMIA, BRONCHITIS, CONJUNCTIVAL CONGESTION, AND SLOW CEREBRATION. IT IS OF INTEREST TO NOTE THAT PRADHAN EXAMINED THE URINE IN ALL CASES, AND FOUND THAT ALBUMINURIA WAS ALWAYS PRESENT EXCEPT IN MILD CASES. IN THE SEVERE CASES A FEW GRANULAR AND BLOOD CASTS WERE SEEN. INCREASING ALBUMINURIA WITH BLOOD CASTS WAS A BAD PROGNOSTIC SIGN. THIS CLINICAL FINDING IS INTERESTING, IN VIEW OF THE PATHOLOGICAL FINDINGS MADE BY ALLEN AND SPITZ (2) IN SCRUB TYPHUS. NO WEIL-FELIX TESTS WERE CARRIED OUT, BUT THE CLINICAL DESCRIPTION AND SEASONAL INCIDENCE SUGGEST
THAT THESE MIGHT HAVE BEEN CASES OF SCRUB TYPHUS. SOME CASES WERE INFESTED WITH LICE; SOME CASES WERE CULTIVATORS, WHO WOULD BE EXPOSED TO THE BITES OF MITES (AND ALSO.Ticks AND OTHER POSSIBLE VECTORS).

IN A PAPER IN 1924 MEGAW (189) HAD GIVEN REASONS WHY TYPHUS WAS NOT DIAGNOSED IN THE TROPICS; ITS SUPPOSED RARITY, THE DIFFICULTY OF DETECTING THE RASH ON PIGMENTED SKINS, THE IMPRESSION THAT TYPHUS OCCURRED IN SEVERE AND FATAL EPIDEMICS, ITS RESEMBLANCE TO TYPHOID WITH OFTEN A RISE IN TITRE IN THE WIDAL TEST, ITS SUPPOSED SUDDEN ONSET AND SUDDEN TERMINATION, AND THE PRESUMPTION THAT TYPHUS FEVERS WERE NOT FOUND AMONGST PEOPLE LEADING A CLEAN AND HEALTHY OUTDOOR LIFE IN THE COUNTRY. ROCKY MOUNTAIN SPOTTED FEVER AND JAPANESE RIVER FEVER WERE PRESUMED TO BE CONFINED TO THEIR OWN AREAS. I FOUND THESE VERY ARGUMENTS ALL BROUGHT FORWARD AGAIN IN 1943 WHEN I BEGAN TO DIAGNOSE TYPHUS LIKE FEVERS IN A MILITARY HOSPITAL IN CALCUTTA.

MEGAW IN 1917, (186) 1921, (187) 1924, (189) 1925 (191) AND IN 1934 (193) HAD SUGGESTED THAT THE DISEASE FROM WHICH HE HIMSELF HAD SUFFERED, McKECHNIE'S CASES FROM THE SAME AREA, CONOR AND BRUCH'S (51) FIEVRE BONTONNEUSE FROM TUNIS, MCNAUGHT'S (183) ANOMALOUS PARATYPHOID FEVERS IN SOUTH AFRICA, THE SPOTTED FEVER OF IDAHO DESCRIBED BY MAXEY (177),
DAVIES AND JOHNSON'S (64) DENGUE-LIKE FEVER OF NIGERIA AND BRILL'S DISEASE (34 & 35) WERE PLACE DISEASES, SPREAD BY TICKS OR FLEAS AND WERE A SUBGROUP OF CLASSICAL TYPHUS FEVER, HAVING NO CONNECTION WITH DIRT, FAMINE OR OVERCROWDING.

BRILL'S DISEASE IS NOW GENERALLY ACCEPTED TO BE A RECRUDESENCE OF EXANTHEMATIC TYPHUS CONTRACTED IN CHILDHOOD. FIEVRE BOUTONNEUSE, THE SPOTTED FEVER OF IDAHO AND THE SOUTH AFRICAN DISEASE OF MCNAUGHT ARE SEROLOGICALLY GROUPED TOGETHER BY FELIX (87).

MEGAW CONSIDERED THAT McKECHNIE'S CASES AND OTHER CASES IN THE SAME AREA HAD CONTRACTED THE DISEASE FROM THE BITE OF A TICK. MEGAW'S (187) CASE AGAINST THE TICK AS A VECTOR OF TYPHUS IN INDIA WAS BASED ON THE FOLLOWING NINE POINTS

(1) HE HIMSELF WAS BITTEN BY A TICK IN THE SUSPECTED AREA, THREE WEEKS BEFORE HIS ATTACK OF TYPHUS; HIS WIFE WAS NOT BITTEN AND DID NOT DEVELOP TYPHUS.

(2) ROCKY MOUNTAIN SPOTTED FEVER WAS KNOWN TO HAVE A TICK VECTOR, AND IS CLINICALLY SOMEWHAT SIMILAR IN ITS Milder FORMS.

(3) THE SITE WHERE McKECHNIE'S CASES OCCURRED FAVOURED THE POSSIBILITY OF TICK BITE AS THEY OCCURRED IN COTTAGES SURROUNDED BY JUNGLE. ONE OF THE CASES HAD BEEN ON A
SHOOT DURING THE POSSIBLE INCUBATION PERIOD.

(4) NO CASES OCCURRED IN THE NEIGHBOURING MILITARY ENCAMPMENT WHICH WAS SURROUNDED BY CULTIVATED LAND.

(5) NO CASES WERE SEEN IN NAJNI TAL, A HIGHLY DEVELOPED HILL STATION. BLEWITT (26) HOWEVER RECORDED CASES THERE LATER.

(6) ALL CASES REPORTED IN INDIA COULD HAVE BEEN BITTEN BY TICKS. (THESE POINTS ONLY SUGGEST THE POSSIBILITY OF THE TICK VECTOR).

(7) THE INCIDENCE WAS DIFFERENT FROM DISEASES KNOWN TO BE CARRIED BY HUMAN LICE.

(8) THE DISEASE WAS CONFINED TO CERTAIN LOCALITIES YEAR AFTER YEAR WHICH WOULD BE CONTRARY TO THE POSSIBILTY OF ITS BEING SPREAD BY HUMAN LICE.

(9) THE FREEDOM OF EUROPEANS FROM LOUSE RELAPSING FEVER.

THE LAST THREE POINTS CERTAINLY DO NOT FAVOUR THE SUGGESTION THAT THE VECTOR WAS THE LOUSE. MEGAW’S (187) INTEREST IN MCKECHNIE’S REPORT, AFTER HIS OWN ATTACK OF TYPHUS, TURNED THE ATTENTION OF MEDICAL MEN TO THE POSSIBILITY OF A TYPHUS-LIKE FEVER IN INDIA WITH A VECTOR OTHER THAN THE LOUSE. MEGAW’S TICK HYPOTHESIS WAS BASED ON ANALOGY. SO FAR EXPERIMENTAL PROOF HAS NOT BEEN FORTHCOMING.

THE ISOLATION OF UNKNOWN STRAINS OF RICKETTSIAE
IS A DIFFICULT PROCEDURE AND BLEWITT'S (26) NEGATIVE INVESTIGATION OF TICKS IN THE KUAMON, EXTENSIVE AS IT WAS DOES NOT, IN THE LIGHT OF PRESENT DAY KNOWLEDGE, EXCLUDE THE TICK AS A POSSIBLE VECTOR. THE MORE OFTEN ONE RE-READS MEGAW'S ARTICLES ON TYPHUS IN INDIA THE MORE ONE IS IMPRESSED BY HIS REASONING, THAT A PRIMA FACIE CASE HAD BEEN MADE AGAINST THE TICK. BLEWITT (26) CONCLUDED HOWEVER THAT THE TYPHUS SEEN IN THE KUAMON WAS EITHER LOUSE OR FLEA BORNE. MITES WERE NOT INVESTIGATED. IT IS INTERESTING TO NOTE THAT MCKECHNIE MENTSIONS AN EPIDEMIC OF RELAPSING FEVER IN THE AREA BEFORE HE SAW HIS CASES. THE LOUSE AND THE TICK ARE THE POSSIBLE VECTORS OF THIS DISEASE. IT IS ALSO INTERESTING TO RECOLLECT THAT EXANTHEMATIC LOUSE BORNE TYPHUS APPARENTLY HAS BEEN DORMANT IN THE WEST OF IRELAND. McCONN (179) HAS DESCRIBED HOW FOR MANY YEARS NO CASES HAD BEEN SEEN. THEN EPIDEMICS OF WHAT HAD BEEN DIAGNOSED AS "MEASLES" WERE FOLLOWED IN SUBSEQUENT YEARS BY CASES OF TYPHUS. WHEN TYPHUS IS NOT EXPECTED CONFUSION IS POSSIBLE. INVESTIGATION REVEALED THAT CERTAIN CASES DIAGNOSED AS 'INFLUENZA' WERE PROBABLY MILD CASES OF TYPHUS. IT DOES NOT SEEM IMPOSSIBLE THAT IN THE KUAMON TYPHUS MAY REMAIN DORMANT IN THE SAME WAY, ONLY BECOMING APPARENT AT CERTAIN TIMES, SUCH AS THE EXPOSURE TO INFECTION OF "SUSCEPTIBLES" FROM
OTHER PLACES. NAPIER (214) HAS MENTIONED HOW THE ARMY IN INDIA WAS ON OCCASIONS SO EXPOSED AND DETECTED LATENT INFECTION, AND THIS POINT HAS BEEN MENTIONED MORE THAN ONCE IN INDIAN MEDICAL GAZETTE EDITORIALS (75 & 76) EXANTHEMATIC LOUSE BORNE TYPHUS IS PRESENT ON THE MOUNTAINOUS FRONTIERS OF INDIA. IT IS POSSIBLY MODIFIED IN SOME INSTANCES. OTHER TYPHUS LIKE FEVERS MAY EXIST. THE TICK HAS MEANTIME NOT BEEN INCRIMINATED AS A VECTOR, BUT HAS NOT BEEN EXCLUDED. THE MITE HAS NOT BEEN INVESTIGATED.

THE FOREGOING CASES IN THE INDIAN LITERATURE CANNOT BE CLASSIFIED WITH ANY DEGREE OF CERTAINTY THEIR TENTATIVE CLASSIFICATION IS PUT FORWARD IN A TABLE USING THE RATHER INDEFINITE CRITERIA MENTIONED PREVIOUSLY.

SEE TABLE TWELVE.

ALSO SEE MAPS

THREE, FOUR AND FIVE

2. THE INDIAN LITERATURE AFTER THE STANDARDIZATION OF THE WEIL-FELIX TEST.

CHRISTIAN (49) DESCRIBED A CASE IN DELHI IN 1932 WHERE BEFORE THE ONSET OF FEVER A TICK WAS DISCOVERED OVER THE INGUINAL REGION OF A PATIENT. THE DEVELOPMENT OF A LOCAL LESION WITH LOCAL ADENOPATHY AND SWELLING OF THE
TABLE TWELVE.

SUGGESTED VECTOR IN CASES REPORTED IN THE INDIAN LITERATURE 1869-1932.

<table>
<thead>
<tr>
<th>LOUSE</th>
<th>FAIRWEATHER</th>
<th>TRANS INDUS REGION</th>
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<tr>
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<td>RAWALPINDI</td>
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<td>CHEVERS</td>
<td>MALABAND</td>
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<td>CALCUTTA</td>
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<td>THOMPSON</td>
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<td>MEERUT</td>
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| IN SOME CASES MITE NOT ABSOLUTELY IMPOSSIBLE. |
|-----|-----------------|
| MEGAW | KUAMON |
| MEGAW | NAGPUR, CARNPORE |
| MEGAW | HYDERBAD, BANGALORE, ANGOLA (ORISSA), AYYAB, NARAIANG (DACCA) |
| MEGAW AND SUNDER RAO | BALACHAT (C.P.) |
| KEATES | (N.W. FRONTIER) MURREE |
| CUNNINGHAM AND THEODORE | (3 CASES) NARASAPATNAM |

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<thead>
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<td>SACHS</td>
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<tr>
<td>LINDERMAN</td>
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<td>CUNNINGHAM AND THEORE</td>
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<td>MACKENZIE</td>
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<td>WILLIAMSON</td>
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| PRADEHAN | BERAR (C.P.) |

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<th>INSUFFICIENT INFORMATION TO FORM ANY OPINION.</th>
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<td>BANNERJI</td>
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<td>LABARNADIE</td>
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<td>MUKERJ</td>
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MAP THREE

MAP SHOWING LOCATION OF CASES REPORTED PRIOR TO 1932 IN WHICH A TICK IS OFTEN POSTULATED AS THE VECTOR. WHILE BY ANALOGY A TICK IS A POSSIBLE VECTOR, OTHER CONSIDERATIONS MAKE ONE LEAVE THE QUESTION OF THE VECTOR AN OPEN ONE.
MAP SHOWING THE LOCATION OF CASES REPORTED PRIOR TO 1932, IN WHICH A TICK IS OFTEN POSTULATED AS THE VECTOR. IN THESE CASES A CENTRIFUGAL DEVELOPMENT OF A RASH SUGGESTS A POSSIBLE RELATIONSHIP WITH ROCKY MOUNTAIN SPOTTED FEVER, AND A TICK MAY BE CONSIDERED A POSSIBILITY.
MAP FIVE

AREA OF BLEWITT'S INVESTIGATIONS IN 1935-36

BANNERTI, 1927, 1937.

CALCUTTA (BARAKPUR)

ALLAHABAD

DARAKI

MUKERJI, 1926-1932.

LABARNADIE, 1932.

NAVAR

DARWAR

JACKSON, 1924.

POONA

PONDICHERRY

NAGPUR

KAUMON

NEPAL

MAP SHOWING LOCATION OF CASES REPORTED PRIOR TO 1932 IN WHICH INFORMATION IS INSUFFICIENT TO FORM ANY OPINION AS TO THE POSSIBLE VECTOR.
AXILLARY GLANDS ON THE SAME SIDE, TOGETHER
WITH A GENERALISED RASH, WAS CONSISTENT WITH
TSUTSUGAMUSHI DISEASE (SCRUB TYPHUS). THE RASH
HOWEVER WAS NOT QUITE TYPICAL OF THAT DISEASE,
CONSISTING OF LARGE MACULO-PAPULAR SPOTS,
OFTEN PETECHIAL, ABOUT ONE QUARTER OF AN INCH
IN DIAMETER. CHRISTIAN THOUGHT THAT THE TICK
IN THIS CASE WAS THE VECTOR. THE WEIL-FELIX
REACTION ON THE EIGHTH DAY OF THE FEVER WAS
1/125 OXK FALLING TO 1/50 ON THE SIXTEENTH
DAY. THIS WAS PERHAPS SUSPICIOUS, BUT DID
NOT UNEQUIVOCALLY SUPPORT THE SCRUB TYPHUS
DIAGNOSIS. AN ATTEMPT WAS MADE TO ISOLATE A
RICKETTSIA, BY INJECTING WHOLE BLOOD INTRA-
PERITONEALLY INTO A GUINEA PIG, BUT AS IS NOW
KNOWN, THE AMOUNT INJECTED WAS INSUFFICIENT.
THE REACTIONS OF THE GUINEA PIGS WERE CONSIS-
TENT WITH A MILD INFECTION, BUT THE TECHNIQUE
OF PASSAGE TO RABBITS OR MICE WAS THEN UNKNOWN,
AND THE RESULTS WERE NEGATIVE. ONE MAY SUSPECT
THAT THE CASE BELONGED TO THE UNDETERMINED
SEROLOGICAL GROUP OF FELIX (87), THOUGH IT IS
NOT POSSIBLE TO EXCLUDE TSUTSUGAMUSHI DISEASE
WITH COMPLETE CERTAINTY.

IN 1935 CHRISTIAN (50) REPORTED SIX MORE
CASES FROM BAREILLY. FOUR OF THESE WERE BRITISH,
TWO OF WHOM DID NOT HAVE A RASH. SERIAL WEIL-
FELIX TESTS WERE CARRIED OUT IN FIVE OF THE
CASES. A SIGNIFICANT RISE IN LOW TITRE TO OX2
AND OX19 WAS NOTED IN TWO BRITISH AND ONE INDIAN CASE, AND TO OX2, OX19 AND OXK IN ONE BRITISH CASE. ONE INDIAN CASE WAS SEROLOGICALLY SCRUB TYPHUS, THE OXK ALONE RISING TO HIGH TITRE (1/10,000). HE HAD MILD BRONCHITIS. THE TWO BRITISH CASES WHICH DID NOT HAVE A RASH WERE SO MILD THAT CHRISTIAN WONDERED WHETHER THESE WERE RELATED TO THE REPORTED CASES OF "TYPHUS INAPPArente" OF THE FRENCH WRITERS.

BLEWITT (25) REVIEWED THIRTEEN CASES SEEN AT AHMEDNAGAR BETWEEN 1931 AND 1933. THE CLINICAL PICTURE HAD NO SIGNIFICANT FEATURES, EXCEPT THAT THERE WERE SOME VERY TOXAEMIC CASES WITH A PROFUSE RASH, AND IN NONE OF THE CASES WAS BRONCHITIS PRESENT. SOME CASES ALSO WERE VERY MILD AND ASKED FOR A FULL DIET DURING THEIR ILLNESS. THE SEROLOGICAL REACTIONS WERE NOT CLEAR CUT, "OX19 WAS USUALLY EARLIEST, HIGHEST AND LONGEST". A PETECHIAL ELEMENT WAS PRESENT IN THE RASH. BLEWITT WAS OF THE OPINION THAT A TICK WAS THE VECTOR. ON THE EVIDENCE THIS IS POSSIBLE, IN SO FAR AS THERE WAS NO OBVIOUS VECTOR. THE CASES OCCURRED DURING THE TRAINING SEASON, WHEN CONTACT WITH OTHER ECTOPARAZITES IN THE JUNGLE WAS POSSIBLE. THESE MILD CASES SEEN BY CHRISTIAN AND BLEWITT ARE OF INTEREST. SEROLOGICALLY CHRISTIAN AND BLEWITT'S CASES WOULD SEEM TO BE IN FELIX'S
UNDETERMINED GROUP WITH THE EXCEPTION OF THE SINGLE CASE OF CHRISTIAN'S WHICH WAS SUGGESTIVE OF SCRUB TYPHUS.

BOYD'S (28) IMPORTANT PAPER APPEARED IN 1935, SUMMARIZING ONE HUNDRED AND EIGHT CASES REPORTED BY THE ARMY IN INDIA IN 1934. MILITARY LABORATORIES WERE NOW SUPPLIED WITH STANDARDIZED ALCOHOLIC PROTEUS SUSPENSIONS BY BRIDGES (31) FROM KASAULI. BOYD CLASSIFIED THE REPORTS ON THE SEROLOGICAL RESULTS. IN NO CASE WAS THERE UNEQUIVOCAL EVIDENCE THAT THE LOUSE WAS THE VECTOR; NOR ONE WOULD EXPECT OF COURSE EXANTHEMATIC LOUSE-BORNE TYPHUS TO BE FOUND IN A MODERN ARMY IN PEACE TIME. HIS CASES THEREFORE, WHILE PROBABLY INCLUDING EXAMPLES OF THE MAIN TYPHUS GROUPS SEEN IN INDIA, DO NOT INCLUDE LOUSE-BORNE TYPHUS. SEROLOGICALLY HE FOUND THAT OXK CASES WERE FAIRLY CLEAR CUT, WITH NO CO-AGGLUTININS. THEY WERE FOUND WIDELY OVER NORTH EASTERN AND SOUTHERN INDIA ROUND POONA, AHMEDNAGAR AND DECCAN. THERE WAS A DEFINITE SEASONAL INCIDENCE IN THE SECOND HALF OF THE YEAR, MAXIMUM IN AUGUST - SEPTEMBER. THE CENTRIPETAL APPEARANCE OF THE MACULOR RASH ON THE TRUNK IN FIFTEEN OUT OF TWENTY-ONE BRITISH SOLDIERS AND ONLY ONE OUT OF FOURTEEN INDIAN SOLDIERS AND THE LACK OF ANY EVIDENCE TO SUGGEST THE VECTOR, IS CHARACTERISTIC OF THE LATER OUTBREAKS DURING
THE WAR IN BURMA AND ASSAM. THE ABSENCE OF ADENITIS AND THE ABSENCE OF A PRIMARY SORE IS CONSISTENT WITH THE DESCRIPTIONS OF CASES SEEN IN CALCUTTA DURING THE SAME PERIOD.

HIS SEROLOGICAL OX2 CASES AND ONE GROUP OF SEROLOGICAL OX19 CASES COME FROM THE AREA ROUND POONA AND THE DECCAN. IN THE OX2 CASES, A MACULAR, THEN PAPULAR AND PETECHIAL RASH WAS PRESENT IN THIRTEEN OUT OF FOURTEEN CASES, AND BEGAN CENTRIFUGALLY BEFORE BECOMING GENERALLY DISTRIBUTED. CO-AGGLUTINATION WAS COMMON.

OTHER CASES FROM THE SAME AREA HAD PREDOMINANT OX19 AGGLUTININS WITH CO-AGGLUTININS FOR OX2 AND OXK. CLINICALLY THEY RESEMBLE THE CASES WITH PREDOMINANT OX2 AGGLUTININS, AND BLEWITT'S (25) CASES SEEN IN THE SAME AREA. SEROLOGICALLY THESE CASES RESEMBLE THOSE DESCRIBED BY HEILIG AND NAIDU (117 & 118) IN MYSORE SOME 470 MILES TO THE SOUTH IN 1941. IN A LATER PAPER IN 1944 HEILIG AND NAIDU (119) CONSIDER THAT THEIR CASES ARE THE SAME AS BOYD'S SEROLOGICAL OX2 GROUP.

BOYD'S OTHER SEROLOGICAL GROUP OF OX19 CASES SEEN MOSTLY IN BANGALORE ARE ALMOST CERTAINLY MURINE TYPHUS AS BOYD SUGGESTS.

BOYD'S PAPER IS IMPORTANT IN THAT IT IS THE ONLY ATTEMPT TO CORRELATE THE TYPHUS FEVERS OF THE WHOLE OF INDIA USING THE WEIL-
FELIX TEST WITH STANDARDIZED SUSPENSIONS AND TECHNIQUE. HE CONSIDERED IN AGREEMENT WITH FELIX (86) THAT AS IN FIEVRE BOUTONNEUSE, THE OX2 AND OX19 AGGLUTININS SEEN IN CASES IN THE POONA, AHMEDNAGAR AREA, ARE OF THE NATURE OF GROUP AGGLUTININS.

SEE MAP SIX.

MARTIN AND ANDERSON (175) WERE THE FIRST TO REPORT A CASE OF SCRUB TYPHUS FROM BURMA IN 1933. THE DISEASE WAS CONTRACTED BY A EUROPEAN IN THE JUNGLE. THERE WAS A PROFUSE RASH. THE PROTEUS OXK SUSPENSIONS WERE NOT OBTAINED UNTIL SOME TIME AFTERWARDS. SERUM TAKEN ON THE EIGHTEENTH DAY OF THE DISEASE HAD BEEN KEPT AND WAS FOUND TO AGGLUTINATE LIVING SUSPENSIONS OF PROTEUS OXK. MARTIN AND ANDERSON THOUGHT THAT THE INFECTION IN THIS CASE, LIKE KUNDU'S CASE FROM RANGOON, HAD BEEN IMPORTED SOMEHOW FROM MALAYA.

MAITRA AND SEN GUPTA (173) HOWEVER, WERE ABLE TO REPORT IN 1936 THAT ROUTINE EXAMINATION OF SERUM SENT FOR THE WIDAL TEST FROM ALL PARTS OF BURMA, REVEALED 109 CASES IN WHICH ON SEROLOGICAL GROUNDS A TENTATIVE DIAGNOSIS OF TYPHUS, EITHER RURAL OR URBAN, COULD BE MADE. ONLY FOUR CASES SHOWED A RASH. TWO SEROLOGICAL VARIETIES, OX19 AND OXK, APPEARED INDISCRIMINATELY IN RURAL AND URBAN AREAS, IN TWENTY-FOUR OUT
MAP SIX.
MAP SHOWING DISTRIBUTION OF BOYD'S CASES.

SEROLOGICAL OXK CASES REPORTED FROM NORTH EASTERN COMMAND SOUTHERN COMMAND EXCEPT AROUND POONA AHMEDNAGAR AND MADRAS.

SEROLOGICAL OX2 CASES REPORTED FROM POONA INDEPENDENT BRIGADE AREA DECCAN

SEROLOGICALLY OX10 CASES PROBABLY MURINE TYPHUS REPORTED FROM POONA AREA AND BANGALORE SOUTHERN COMMAND
OF FORTY DISTRICTS. THREE CASES AGGLUTINATED OX2 MAINLY, ONE CASE BEING IN RANGOON, AND TWO IN HENZADA ON THE IRRAWADDY. NO ESCHARS WERE REPORTED. THE POSSIBILITY OF THEIR PRESENCE IS NOT EXCLUDED HOWEVER.


WILSON (340) IN 1935 REPORTED THE FIRST SEROLOGICALLY PROVED CASE OF OXK TROPICAL TYPHUS CONTRACTED IN CALCUTTA. THE PATIENT REPORTED SICK ON HIS ARRIVAL IN BURMA; THE JOURNEY BY SEA FROM CALCUTTA TO RANGOON TAKES ABOUT THREE DAYS. WILSON RECOGNISED THAT THE DISEASE SEROLOGICALLY AND CLINICALLY RESEMBLED THE SCRUB TYPHUS OF MALAYA BUT REMARKED THAT "THE VICINITY OF CALCUTTA WOULD NOT APPEAR TO BE THE SORT OF PLACE ONE WOULD ASSOCIATE WITH SCRUB TYPHUS", SHOWING HOW LABELS FOR DISEASES MAY BE UNSUITABLE. IN FACT THE 'MAIDAN' (PARADE GROUND) OR PARK SURROUNDING FORT WILLIAM WHERE THE PATIENT WAS STATIONED IN CALCUTTA, IS QUITE LARGE
ENOUGH TO HARBOUR MANY VARIETIES OF RODENTS. IT ALSO ADJOINS THE JETTYS ON THE HOOGLI RIVER AT ONE POINT.

STOTT (303) IN 1935 REPORTED TWO CASES WHO CONTRACTED TYPHUS-LIKE FEVER IN HAMIRPUR, IN THE UNITED PROVINCES, WHILE TOURING IN THE JUNGLE, ONE WAS SEROLOGICALLY OX.19. INOCULATION OF A GUINEA PIG WITH 4 C.C. OF BLOOD INTRAPERITONEALLY WAS PROBABLY CARRIED OUT TOO LATE IN THE DISEASE TO HAVE ANY CHANCE OF SUCCESS. THE GUINEA PIG REACTED WITH LOSS OF WEIGHT ONLY. STOTT WAS ABLE TO SHOW THAT THE WASSERMANN REACTION WAS POSITIVE DURING THE DISEASE, TO BECOME VERY LESS STRONGLY POSITIVE, AND THEN NEGATIVE IN THREE WEEKS. HE ALSO NOTED THE RISE AND RAPID FALL IN TYPHUS-LIKE FEVER OF TYPHOID 'H' AGGLUTININS IN AN UNINOCULATED PERSON. STOTT CONSIDERED A TICK TO BE VECTOR IN HIS TWO CASES AS THEY HAD BEEN IN PARTS OF THE JUNGLE IN WHICH TICKS WERE NUMEROUS. THE HIGH TITRE OF OX.19 IN ONE CASE IS NOT PARTICULARLY HELPFUL. SEROLOGICALLY IT SUGGESTS MURINE TYPHUS.

BUSH (41) IN 1936 REPORTED SIX CASES IN FOUR BRITISH AND TWO INDIAN SOLDIERS, CONTRACTED IN THE OPEN COUNTRY IN THE SIMLA HILLS, TOWARDS THE END OF THE RAINS. SEROLOGICALLY THESE CASES WERE NOT ALL ALIKE, ONE CASE HAVING AGGLUTININS FOR OX2 AND OX19, THE OTHERS BEING CLEAR CUT
OXK, SUGGESTIVE OF SCRUB TYPHUS.

MACNAMARA (167) IN 1935 REPORTED SIXTEEN CASES OF TYPHUS-LIKE FEVER, SEROLOGICALLY OXK FROM THE SIMLA HILLS. CLINICALLY THE DESCRIPTION WAS SUGGESTIVE OF SCRUB TYPHUS. THERE WAS ONE FATAL CASE WITH INCONCLUSIVE SEROLOGICAL RESULTS.

COVELL (54) IN SIMLA WAS ABLE TO ISOLATE A MURINE STRAIN FROM LOCAL RATS BUT NOT FROM THE RECENTLY IMPORTED GREY PALM SQUIRRELS. COVELL AND MEHTA (57) WERE ABLE TO TRANSMIT AN OX19 STRAIN FROM RAT BRAIN TO LICE. BUT ATTEMPTS TO INFECT MONKEYS WITH THE INFECTED LICE WERE INCONCLUSIVE. ATTEMPTS TO ISOLATE A 'K' STRAIN FROM RATS OR SQUIRRELS WERE UNSUCCESSFUL. WEBSTER (326) IN 1940 REPORTED THAT THE LARVAL TROMBICULA MITE, T. DELIENSIS THEN SUSPECTED ON VERY STRONG GROUNDS TO BE THE VECTOR OF SCRUB TYPHUS IN MALAYA WAS PRESENT ON THE LOCAL BROWN MONKEY (SILENUS (MACCAUS) Rhesus). ATTEMPTS TO ISOLATE A STRAIN FROM THE MONKEYS OR THE MITES WAS UNSUCCESSFUL. ATTEMPTS TO INFECT THE MONKEYS WITH LOCAL HUMAN 'K' RICKETTSIAL STRAINS WAS UNSUCCESSFUL. WEBSTER REPORTS THE DIFFICULTY OF ISOLATING 'K' STRAINS FROM THE RATS Owing TO FREQUENT CO-INCIDENT INFECTION WITH MURINE STRAINS AND SPIRILLUM MINUS.

COVELL (53) FOUND THAT IN TRAPPED WILD
RATS, 2.1% HAD A POSITIVE WEIL-FELIX OF 1/250 OXK, WHICH HE CONSIDERED TO BE SUGGESTIVE OF
TYPHUS INFECTION. THIS CONCLUSION HOWEVER, IS
NOW CONSIDERED TO BE OPEN TO DOUBT. SAVOOR
AND LEWTHWAITE (274) HAVE POINTED OUT THAT RATS
COMMONLY HARBOUR SPIRILLUM MINUS, WHICH CAN
PRODUCE A POSITIVE AGGLUTINATION OF PROTEUS
OXK. BRIGHAM AND BENGSTON (33) HAVE SHOWN
THAT IN MURINE TYPHUS THE WEIL-FELIX TEST MAY
GIVE BOTH FALSE POSITIVE AND FALSE NEGATIVE
RESULTS.

VAUCEL AND BRUNEAU (316) HAVE CULTIVATED BOTH 'H' AND 'O' VARIANTS OF PROTEUS
FROM RATS, INCLUDING A STRAIN CULTURALLY AND
SEROLOGICALLY IDENTICAL WITH PROTEUS OXK.

MEHTA (199) REPORTED IN 1937 THE
RESULTS OF INVESTIGATION OF THE ECTO-
PARAZITES FOUND ON 2,451 RATS AND SHREWS
TRAPPED IN KASAULI AND SABATHU. LICE AND
TICKS WERE NOT CONSIDERED TO BE VECTORS OF
X19 TYPHUS, BUT TICKS WERE CONSIDERED
POSSIBLE VECTORS OF OXK TYPHUS. TROMBICULA
DELIENSIS WAS FOUND THROUGHOUT THE YEAR, AND
WAS PARTICULARLY ABUNDANT DURING THE SEASON
WHEN XX TYPHUS WAS ENCOUNTERED IN THOSE
AREAS.

FURTHER WORK IS REQUIRED, BEFORE IT
MAY BE SAID TO BE PROVED THAT IN INDIA PROPER,
THE SCRUB TYPHUS RICKETTSIAE HAVE THE FIELD RAT AS A HOST AND A LARVAL MITE AS A VECTOR.

SHORTT AND D’SILVA (286) WHOSE ARTICLE ON THE DISTRIBUTION OF TYPHUS-LIKE CASES (WHICH THEY REFER TO AS INDIA TICK TYPHUS), HAS BEEN MENTIONED WHEN DISCUSSING MEGAW’S HYPOTHESIS THAT THE TICK WAS THE VECTOR OF ENDEMIC TYPHUS IN INDIA, HAD COLLECTED NOTES ON ONE HUNDRED AND SEVENTY-SEVEN CASES OF THE DISEASE IN INDIA UP TO 1936. THEY FOUND THAT THERE WAS A GENERAL TENDENCY FOR CASES OF THE TYPHUS GROUP OF FEVERS TO OCCUR IN THE SUMMER IN THE ELEVATED PART OF THE COUNTRY (BETWEEN 4500 AND 6000 FEET) AND IN THE WINTER SEASON IN THE PLAINS. SUCH AN INVESTIGATION, IF MADE WITH THE INTENTION OF FINDING A CLUE TO THE VECTOR, NOT SURPRISINGLY DID NOT PROVIDE AN OBVIOUS POINTER. SHORTT CARRIED OUT SOME INCONCLUSIVE ANIMAL EXPERIMENTS.

BLEWITT’S (26) INVESTIGATION IN THE KUMAON (OR KUMAUM) HILLS REPORTED IN 1936 HAS ALREADY BEEN MENTIONED IN CONNECTION WITH MEGAW’S TICK HYPOTHESIS. IT WAS THE OUTCOME OF THE SUGGESTION MADE BY McKECHNIE (181) IN HIS REPORT IN 1913. "THE CONCLUSION", SAYS McKECHNIE, "THAT 'JHAR' IS TYPHUS, IS BASED ON EXCLUSION, CLINICAL OBSERVATION AND INDUCTIVE REASONING. IT REQUIRES THE FURTHER SUPPORT OF THE EXPERIMENTAL METHOD". BLEWITT
FAILED TO ISOLATE ANY RICKETTSIAE FROM TICKS COLLECTED IN THE AREA. THERE WERE NO CASES OF TYPHUS-LIKE FEVER TO INVESTIGATE WHILE HE WAS THERE. ONLY SPORADIC CASES WERE SEEN AFTER 1913, THOUGH THE INFECTION DID NOT APPEAR TO HAVE LOST ITS VIRULENCE. McCONN (179) HAS REPORTED OUTBREAKS OF CLASSICAL TYPHUS IN THE WEST OF IRELAND WHERE IT HAS NOT BEEN SEEN FOR MANY YEARS. BLEWITT CONCLUDED THAT TYPHUS WAS ENDEMIC IN THE KUMAON THAT IT WAS OF THE 'HUMANIZED' LOUSE BORNE TYPE, THOUGH THE FLEA BORNE MURINE TYPHUS WAS POSSIBLY ALSO PRESENT. HE DID NOT THINK THE TICK WAS A VECTOR.

CURRAN (61) IN 1936 DESCRIBED TEN CASES SEEN DURING THE YEAR AT JUBBLEPORE. THESE CASES CONFORMED TO A CLINICAL TYPE WITH VERY SEVERE CONSTITUTIONAL DISTURBANCES. IN ONE CASE THE WEIL-FELIX TITRE FOR OX2 ROSE FROM 1/50 ON THE NINTH DAY TO 1/2500 ON THE FIFTEENTH DAY. IN THE OTHER CASES THERE WAS A RISE IN TITRE TO ALL THREE SUSPENSIONS. THE RASH BEGAN CENTRIFUGALLY. THESE CASES HAVE A DISTINCT RESEMBLANCE TO SACH'S (270) CASES AND ALSO TO BLEWITT'S (25) EXCEPT THAT IN THEIR CASES THE RASH DID NOT BEGIN CENTRIFUGALLY AND BLEWITT'S CASES DID NOT HAVE BRONCHITIS AND SOME WERE Milder. THE CENTRIFUGAL APPEARANCE OF THE RASH IN
CURRAN'S CASES AND THE RESPONSE TO ALL THREE PROTEUS SUSPENSIONS RESEMBLE THE CASES IN HEILIG AND NAIDU'S SERIES (117 & 118) WHOSE PATIENTS ALSO DID NOT HAVE BRONCHITIS. THE SEROLOGICAL RESULTS WERE GIVEN IN DETAIL IN TWO CASES.

PANDALAI (235) IN 1936 REPORTED TWO CASES DIAGNOSED AS ENTERIC FEVER IN VIZAGAPATAM IN WHICH THE WEIL-FELIX TEST WITH ALCOHOLIZED SUSPENSIONS GAVE READINGS OF 1/640 AND 1/320 OXK IN TWO CASES. IN NEITHER CASE WAS A RASH SEEN.

BEVERIDGE AND UNDERHILL (20) WHEN REPORTING A CASE OF TSUTSUGAMUSHI FROM SINGAPORE IN 1936, MENTION IN THEIR DISCUSSION A CASE SEEN BY ONE OF THEM IN CENTRAL INDIA. NO CASES HAD BEEN DESCRIBED IN THE NEIGHBOURHOOD BUT SOME OF MEGAW'S CASES WERE FROM AN ADJACENT DISTRICT. THE PATIENT HAD BEEN IN THE JUNGLE, A MONTH BEFORE, AND FOUR DAYS BEFORE HIS ILLNESS AND HAD WALKED THROUGH LONG GRASS AND SCRUB. THE CLINICAL DESCRIPTION OF THE DISEASE AND THE RASH WERE SUGGESTIVE OF SCRUB TYPHUS. THE WEIL-FELIX RESULTS HOWEVER SHOWED CROSS AGGLUTINATION IN LOW TITRE, AND SUGGESTED RATHER THAT THE CASE BELONGED TO THE UNDETERMINED GROUP. IT IS INTERESTING TO KNOW THAT A COMRADE NOTED TICKS UPON THE PATIENT THOUGH AS FAR AS HE
KNEW HE WAS NOT BITTEN.

KAPILA AND MAITRA (129) IN 1937 REPORTED A SEVERE CASE OF SCRUB TYPHUS FROM BHAMO IN BURMA, NEAR THE CHINESE BORDER. THIS CASE IN SPITE OF THE RASH APPEARING CENTRIFUGALLY, MAY HAVE BEEN A TRUE CASE OF SCRUB TYPHUS, BEING SEROLOGICALLY OXK 1/770. OX2 WAS POSITIVE 1/28 WHICH IS NOT SIGNIFICANT. MAITRA NOTED THE SUBSTANTIAL STIMULATION OF THE TYPHOID 'H' AGGLUTININS IN A RECENTLY INOCULATED SUBJECT, AND THE FALL OF TYPHOID 'O' AGGLUTININS FROM A RAISED TITRE. MAITRA HAD SEEN SIMILAR TRIVIAL STIMULATION OF TYPHOID 'O' AGGLUTININS IN CASES OF TYPHUS AMONGST THE POLICE AND PRISON PERSONNEL WHO HAD BEEN GIVEN PROTECTIVE VACCINE INOCULATION AGAINST TYPHOID FEVER.

COVELL'S (52) SUMMARY OF TYPHUS IN INDIA PUBLISHED IN 1936 HAS BEEN REFERRED TO. ALSO THE OBSERVATIONS OF HIMSELF AND HIS COLLABORATORS ON THE WEIL-FELIX TESTS CARRIED OUT ON THE KASAULI WILD RATS AND STUDIES OF POSSIBLE HOSTS AND VECTORS. WHAT WAS APPARENTLY A MURINE STRAIN WAS RECOVERED FROM WILD RATS. THOUGH THE SEROLOGICAL RESULTS IN ONE GUINEA PIG SUGGESTED TO COVELL THE POSSIBILITY OF A MIXED OX19 AND OXK INFECTION, NO OXK STRAIN COULD BE ESTABLISHED. COVELL AND MEHTA (55) ESTABLISHED A STRAIN FROM RAT FLEAS,
WHICH CROSS IMMUNIZED WITH THE STRAIN FROM RAT BRAINS. THUS, ALTHOUGH SCRUB TYPHUS WAS BEING DIAGNOSED ON CLINICAL AND SEROLOGICAL GROUNDS AT KASAULI, AN OXK STRAIN COULD NOT BE ISOLATED THOUGH RABBITS WERE USED AS WELL AS GUINEA PIGS. LEWTHWAITE AND SAVOOR (148) AND ANIGSTEIN (5) HAD CONSIDERABLE DIFFICULTY IN ISOLATING XK STRAINS IN MALAYA USING GUINEA PIGS ONLY.

YACOB (349) IN 1937 REPORTED TWO SMALL OUTBREAKS OF SEVEN CASES AND FOUR CASES, IN THE PUNJAB NEAR THE INDUS RIVER. THE CASES WERE NOT PROVED SEROLOGICALLY; THE WEIL-FELIX CARRIED OUT IN CONVALESCENCE WAS IN MANY CASES ONLY 1/30 OX19.

SARKAR (272) IN 1939 REPORTED A CASE SEEN 60 MILES FROM RAIPUR IN THE CENTRAL PROVINCES. THERE WAS A RISING TITRE TO OX2 ALONE. THERE WAS NO PARTICULAR DESCRIPTION OF THE RASH. BRONCHITIS WAS NOTED TO BE PRESENT. THIS CASE MIGHT BE GROUPED WITH BOYD'S OX2 CASES.

WEBSTER (326) IN 1940 PUBLISHED FURTHER REPORTS ON INVESTIGATIONS ON TYPHUS IN THE SIMLA HILLS. XK STRAINS HAD BEEN ISOLATED FROM HUMAN CASES OF TYPHUS, BUT NOT FROM ANY POSSIBLE ANIMAL RESERVOIR OR POSSIBLE ARTHROPOD VECTOR, IN PARTICULAR FROM RATS OR TROM-BICULID MITES.
SMITH AND MEHTA (291), also in Simla, apparently recovered an OXK strain from a
Trombicula infested rat, but the strain could not be established owing, it was considered,
to co-incident X19 typhus, and Spirillum minus infection. Evidence was sought, to prove that
the brown monkey (Silenus (Macaus) Rheus) was
the reservoir of XK typhus, as it naturally
habours Trombicula Deliensis. Attempts to
isolate strains from both the monkeys and the
mites failed. Artificial infection of Simla
Hills wild rats (Rattus Rattus) with human
XK strains was rapidly fatal. White rats
when infected got an 'infection inapparente'.
Stray dogs showed no evidence of rickettsial
infection, neither did bed bugs (which had
fed on OXK infected cases).

WEBSTER was not satisfied that the
monkey was eliminated as a possible reser-
voir of typhus. He considered that there
might be an unknown arthropod vector acting
as the reservoir for human XK typhus between
seasons.

MAKIE (169) was to demonstrate later
the trans-ovarial transmission of R. orientalis
in Trombicula Deliensis Walch in Assam.

PATEL (243) in April 1940 and (244) in
November 1940 reported seven and eight cases
from Bombay City. Amongst the first seven
CASES, THE DIAGNOSIS OF ONE WAS DOUBTFUL. IN NO CASE WAS A RASH SEEN. THE AVERAGE SHORTER DURATION (OF ABOUT EIGHT DAYS) AND THE POSITIVE WEIL-FELIX TO OX19 SUGGESTED THAT THESE WERE CASES OF MURINE TYPHUS.

MINCHIN (201) REPORTED AN OBSERVATION ON THE PLATELET COUNT IN A CASE OF TYPHUS WITH HAEMATURIA. THE COUNT WAS FOUND TO BE BELOW THE CRITICAL LEVEL, AND ROSE WITH IMPROVEMENT IN THE CLINICAL CONDITION.

SHARMA (285) PUBLISHED IN 1940 A SUMMARY OF FIFTY-SIX CASES SEEN IN BANGALORE - A HILL STATION NEAR MYSORE. THE CLINICAL DIAGNOSIS WAS MADE ON FAIRLY ADEQUATE CRITERIA. A RASH WAS TYPICALLY SEEN IN ONLY TWELVE CASES. IN THE OTHER CASES IT WAS DIFFICULT TO SEE. ANGLO-INDIANS (EURASIANS), WHO ARE THE OFFSPRING OF THE UNION BETWEEN EUROPEANS AND SOUTHERN INDIANS, MAY HAVE VERY DARK SKINS. MOST OF SHARMA'S CASES WERE OF THAT COMMUNITY. THE WEIL-FELIX WAS USUALLY ONLY CARRIED OUT ONCE IN EACH CASE AND A TITRE OF 1/125 WAS ACCEPTED AS DIAGNOSTIC. THE RASH WAS PROMINENT ON THE TRUNK. MOST CASES WERE SEROLOGICALLY OX19. ONLY ONE IMPORTED CASE WAS OXK. WITH THIS EXCEPTION THE DESCRIPTIONS ARE CONSISTENT WITH A DIAGNOSIS OF MURINE TYPHUS.

GOYAL (105) PUBLISHED IN 1941 A NOTE
ON A RICKETTSIAL EPIZOOTIC IN CALCUTTA WILD RATS. GOYAL'S INVESTIGATIONS SHOWED THAT THE EPIZOOTIC WAS PRESENT FROM JUNE 1937 TO DECEMBER 1938. THEREAFTER IT APPARENTLY DIED OUT. TWO VOLUNTEERS WERE INOCULATED WITH RICKETTSIAE ISOLATED FROM THE RATS, BUT ONE SHOWED NO SIGNS OF INFECTION AFTER THREE MONTHS. IN THE OTHER CASE THE MAN WAS LOST SIGHT OF AFTER SEVEN DAYS, DURING WHICH TIME HE HAD HAD NO SYMPTOMS.

WOODHEAD AND DUTTA (343) IN 1941 REPORTED THE RESULTS OF COINCIDENTAL WEIL-FELIX TESTS CARRIED OUT ON THE SERA OF 203 CASES SENT FOR THE WIDAL TEST IN ASSAM. STANDARD AGGLUTINATION OF 1/150 OXK OR 1/125 OX19 WITH A SUGGESTIVE HISTORY AND NEGATIVE WIDALS WAS CONSIDERED DIAGNOSTIC. IT IS A LITTLE DIFFICULT TO SAY WHETHER THESE CRITERIA WERE SUFFICIENT TO ENABLE A DIAGNOSIS OF TYPHUS TO BE MADE IN ALL CASES. SEVENTEEN CASES WERE CONSIDERED TO BE FEVERS OF THE TYPHUS GROUP. ON THE WEIL-FELIX RESULTS SIX WERE CONSIDERED TO BE MURINE TYPHUS, THREE TICK TYPHUS AND EIGHT SCRUB TYPHUS. IT IS NOT CLEAR JUST HOW THIS DIFFERENTIATION WAS MADE.

GURBUKSH SINGH (109) REPORTED IN 1945 AN EPIDEMIC OF ONE HUNDRED AND SEVEN CASES SEEN IN SEPTEMBER 1941. THOUGH IT WAS NOT
MENTIONED IN THE REPORT FOR SECURITY REASONS, THE CASES WERE SEEN NEAR MANDALAY IN BURMA. AN ESCHAR WAS SEEN IN THIRTY-EIGHT CASES. LYMPHADENITIS WAS NOTED IN EIGHTY-EIGHT CASES. A RASH WAS SEEN, MAINLY ON THE TRUNK IN THIRTY-EIGHT CASES. THE RASH WAS MACULAR IN THIRTY CASES, PAPULAR IN EIGHT. THESE SIGNS AND THE SPLEENOMEGALY SEEN IN TWENTY-NINE CASES, CONJUNCTIVAL INJECTION, VOMITING AND PAIN IN THE ABDOMEN WAS CONSISTENT WITH A TYPHUS INFECTION. CONVALESCENCE WAS SLOW. THERE WERE ONLY TWO DEATHS. GURBUKSH SINGH CONSIDERED THAT THE EPIDEMIC WAS POSSIBLY LOUSE-BORNE. THE EPIDEMIC WAS LIMITED TO ONE LOUSE INFESTED UNIT, AND IN THAT UNIT A LOUSE FREE COMPANY WERE FREE FROM THE DISEASE. THE OFFICERS WHO SLEPT IN COTS ESCAPED INFECTION. THE MEN INFECTED SLEPT ON THE GROUND. AS THE EPIDEMIC OCCURRED AT THE END OF THE RAINS IT SEEMS POSSIBLE TO ASSUME THAT THE MITES IN THE SOIL COULD HAVE BEEN THE VECTORS. THE ESCHAR AND LYMPHADENITIS IS HIGHLY SUGGESTIVE OF SCRUB TYPHUS. SEROLOGICALLY HOWEVER THE RESULTS WERE MAINLY, BUT NOT ALL, CLEAR CUT OXK. IN SIXTEEN CASES THERE WAS CROSS AGGLUTINATION WITH OX19 AND IN ELEVEN CASES WITH OX2, A SOMewhat SIMILAR FINDING TO SOME OF THE CASES IN MY OWN SERIES IN CALCUTTA.
HEILIG AND NAIDU (117) IN 1941 REPORTED THEIR FIRST FOUR CASES, AND IN 1942 A FURTHER TEN CASES (118) FROM MYSORE. THESE CASES ARE OF EXTREME INTEREST AS THEY ARE OF A GROUP OF INDETERMINED VECTOR, AND WERE MORE THOROUGHLY INVESTIGATED THAN MOST INDIAN CASES. LATER INVESTIGATIONS BY TOPPING (119) WITH THE COMPLIMENT FIXATION TEST WERE TO SHOW A CLOSE CONNECTION WITH ROCKY MOUNTAIN SPOTTED FEVER. CLINICALLY THESE CASES HAD A SUDDEN ONSET, A REMITTENT FEVER FOR TWO OR THREE WEEKS, CONJUNCTIVAL CONGESTION, OCCASIONAL SLIGHT SPLENIC ENLARGEMENT AND A RASH WHICH WAS PROMINENT ON THE WRISTS, PALMS AND SOLES THOUGH APPEARING LATER ON THE BODY. THE WEIL-FELIX IN THE FIRST FOUR CASES WAS NEGATIVE. THE LATER CASES GAVE HIGH TITRES IN SOME CASES TO OX2 WITH FREQUENTLY CO-AGGLUTININS FOR OX19 AND OXK. THE NEIL MOOSER SCROTAL REACTION WAS POSITIVE IN SOME CASES, AND NEGATIVE IN OTHERS. THE INFECTION WAS PASSAGED IN GUINEA PIGS WITH THE DEMONSTRATION OF RICKETTSIAE. THERE WAS A SEASONAL INCIDENCE MAXIMAL IN AUGUST. ALL THE CASES WERE IN CONTACT WITH RATS. ATTEMPTS WERE MADE TO ISOLATE TYPHUS INFECTION FROM RAT FLEAS, AND FROM CATTLE TICKS, WITH WHICH THE PATIENTS WERE ALSO IN CLOSE CONTACT. THE RESULTS WERE INCONCLUSIVE. THE EXPERIMENTS
WERE NOT EXTENSIVE. HEILIG AND NAIDU CONCLUDED THAT NEITHER LICE, TICKS NOR MITES WERE THE VECTORS. THEY COMPARED THEIR CASES WITH BOYD'S POONA, AHMEDNAGAR, JUBBLEPORE, DECCAN GROUP WITH A SIMILAR CLINICAL PICTURE, PREDOMINANT OX2 AGGLUTININS AND CO-AGGLUTININS TO OX19 AND OXK. THEY CONSIDERED THAT THEIR CASES DIFFERED FROM BOYD'S BANGALORE TYPHUS, AND SHERMA'S BANGALORE CASES.

HEILIG AND NAIDU (119) IN 1944 REPORTED A FURTHER EIGHTEEN CASES AND SUMMARIZED THEIR THIRTY-TWO CASES. A FATAL CASE WAS REPORTED, THE WEIL-FELIX OF WHICH WAS DISTINCT FROM THE OTHERS, RISING FROM A TITRE OF OX19 1/280 TO 1/20,000 ON THE THIRTEENTH DAY WITH NO CO-AGGLUTININS. THIS SUGGESTED A POSSIBLE LOUSE OR FLEA-BORNE TYPHUS. ONE CANNOT HELP FEELING THAT ALL THESE CASES, WITH THE ONE EXCEPTION MENTIONED ABOVE, WERE CASES OF THE SAME FORM OF THE DISEASE, FROM THEIR CLINICAL APPEARANCE, SEROLOGY, AND LOCATION. THE VARYING SCROTAL REACTION MIGHT HOWEVER BE HELD TO IMPLY A VARIATION IN THE CASES. BUT ALTHOUGH A HIGHLY INFLAMED TUNICA IS FOUND CONSTANTLY IN MURINE TYPHUS AND ROCKY MOUNTAIN SPOTTED FEVER, ACCORDING TO FINDLAY (93) ONLY A SLIGHT SCROTAL REACTION IS FOUND IN THE MEXICO AND CHINA STRAINS WHICH ARE INTERMEDIATE BETWEEN CLASSICAL EXANTHEMATIC TYPHUS
AND MURINE TYPHUS. KONWENAAR AND WOOLF (141) CITE VARYING DEGREES OF SCROTAL AND TESTICULAR REACTION IN SUMATRAN MITE FEVER, TROPICAL SCRUB TYPHUS AND FIEVRE BOUTONNEUSE. THEY FOUND A NEGATIVE REACTION IN TSUTSUGAMUSHI DISEASE FROM JAPAN AND ANNAM. IT CANNOT THEREFORE BE SAID TO BE AN ABSOLUTE DIAGNOSTIC AID IN THE DETERMINATION OF THE GROUP OF A TYPHUS-LIKE DISEASE.

THE COMPLIMENT FIXATION TESTS CARRIED OUT BY TOPPING WERE WITH THE SERA FROM THREE CASES SENT BY HEILIG AND NAIDU (119). IN ONE CASE THE SCROTAL REACTION HAD BEEN POSITIVE, IN TWO NEGATIVE. TOPPING FOUND THAT THE SERA FIXED COMPLIMENT TO A HIGHER TITRE WITH RICKETTSIAL ANTIGEN FROM ROCKY MOUNTAIN SPOTTED FEVER, THAN WITH A RICKETTSIAL ANTIGEN FROM EITHER EPIDEMIC (CLASSICAL EXANTHEMATIC), OR ENDEMIC (MURINE) TYPHUS. THERE WAS HOWEVER SOME CROSS FIXATION WITH ENDEMIC (OR MURINE) TYPHUS. THE CROSS FIXATION WAS UNEXPLAINED BY TOPPING.

CASTANEDA (44) INTRODUCED THE COMPLIMENT FIXATION TEST TO DIFFERENTIATE MEXICAN TYPHUS FROM BRILLS DISEASE. ITS SPECIFICITY IN AMERICAN ENDEMIC (MURINE) TYPHUS WAS CONFIRMED BY BENGSTON AND TOPPING (19) AND FOR DIFFERENTIATING ROCKY MOUNTAIN SPOTTED FEVER AND BRILLS DISEASE BY PLOTZ AND WERTMAN (254).
PLOTZ AND WERTMAN (255) STUDIED THE MODIFICATION OF THE COMPLIMENT FIXATION RESULTS IN INDIVIDUALS WITH MURINE TYPHUS WHO HAD BEEN GIVEN PROTECTIVE INOCULATION WITH EPIDEMIC TYPHUS VACCINE. THERE WAS A CONSIDERABLE AMOUNT OF CROSS FIXATION; USUALLY THE HIGHER TITRES WERE FOR EPIDEMIC ANTIGEN. THERE WAS CONSIDERABLE CROSS AGGLUTINATION IN THE RICKETTSIAL AGGLUTINATION REACTION, BUT THE WEIL-FELIX REACTION WAS NOT AFFECTED.

BENGSTON AND TOPPING (19) FOUND THAT IN A LABORATORY INFECTION OF FIFTY-THREE CASES WITH ENDEMIC (MURINE) TYPHUS, THE COMPLIMENT FIXATION TEST BECAME POSITIVE ON THE TENTH DAY AND REMAINED POSITIVE FOR FIVE AND A HALF YEARS.

THE COMPLIMENT FIXATION RESULTS IN HEILIG AND NAIDU'S CASES ARE MOST INTERESTING: THEY WOULD SEEM TO BE CONSISTENT WITH A PREVIOUS MURINE INFECTION, POSSIBLY 'INAPPARENTE', THOUGH OF COURSE NOT DIAGNOSTIC OF SUCH AN INFECTION. FURTHER INVESTIGATIONS MIGHT BE ILLUMINATING.

FROM BURMA IN 1942, SONI (294) REPORTED THREE CASES SEEN FROM 1935 ONWARDS; THE FIRST CASE HAD FOR THREE DAYS PARTICIPATED IN AN INTENSIVE RAT DRIVE TEN DAYS BEFORE HIS FEVER BEGAN. A SINGLE WEIL-FELIX WAS CARRIED OUT ON THE EIGHTEENTH DAY OF THE
DISEASE, AND WAS POSITIVE 1/340 TO OX19 WITH LOW CROSS AGGLUTINATION. THIS WAS POSSIBLY A CASE OF MURINE TYPHUS, THOUGH NO RASH WAS SEEN. A SECOND CASE WAS A FEMALE WHO HAD ABORTED. A RASH WAS SEEN ON THE SIXTH DAY OF A FEVER. A SINGLE WEIL-FELIX TEST REVEALED OX19 AGGLUTININS 1/300. THIS IS HARDLY SUFFICIENT TO DIAGNOSE TYPHUS IN VIEW OF POSSIBLE PUERPERAL SEPSIS. A THIRD CASE SHOWED AGGLUTININS FOR BOTH OX2 AND OX19, 1/750 FOR THE FORMER, AND 1/375 FOR THE LATTER. THIS THIRD CASE MIGHT BE A CASE OF TYPHUS-LIKE FEVER OF INDEFINITE WEIL-FELIX GROUP, WHERE THE VECTOR IS UNKNOWN.

BALBIR SINGH (13) IN 1944 REPORTED THIRTY CASES OF TYPHUS FEVER SEEN WITH 'PAI' FORCE. PAI FORCE WERE THE TROOPS IN PERSIA AND IRAQ. BALBIR SINGH CONSIDERED THESE CASES TO BE LOUSE-BORNE, AND CONSIDERED THAT THE INFECTION HAD BEEN INTRODUCED BY REFUGEES FROM POLAND, THE BALTIC AND THE UKRAINE. THREE CASES WERE FATAL. IT IS INTERESTING TO NOTE THAT THESE CASES FREQUENTLY SHOWED CO-AGGLUTININS IN HIGH TITRE TO OXK. IT WOULD SEEM DOUBTFUL WHETHER THESE CASES WERE REALLY IMPORTED FROM EUROPE IN VIEW OF THE CROSS AGGLUTINATION. FROM THE SEROLOGICAL RESULTS THESE CASES ARE OF THE INDETERMINATE GROUP. IN ONE FATAL CASE WHO DIED ON THE
THIRTY-NINTH DAY OF HIS ILLNESS, TUBERCULOSIS, A PELVIC ABSCESS AND A LUNG ABSCESS FOUND AT AUTOPSY RAISE CONSIDERABLE DOUBT WHETHER TYPHUS WAS ALSO PRESENT.

BARDHAN (16) REPORTED IN 1944 FORTY-ONE CASES SEEN IN ARMY PERSONNEL IN THE UNITED PROVINCES OF INDIA. ELEVEN CASES WERE SEEN IN THE SAME RANGE OF HILLS IN WHICH McKECHNIE AND MEGAW SAW THEIR CASES. THERE WAS A CONSPICUOUS ABSENCE OF A RASH IN ALL CASES. THE CASES WERE REPORTED THROUGHOUT THE YEAR INCLUDING THE DRY AND HOT SUMMER MONTHS IN THE PLAINS. CLINICALLY THERE WAS A CLOSE RESEMBLANCE TO "ANY ACUTE UPPER RESPIRATORY INFECTION OF INFLUENZAL TYPE". PHYSICAL SIGNS (MOIST RALES WITH IMPAIRED AIR ENTRY) CONSIDERED TO BE DUE TO "PNEUMONITIS" WERE SEEN IN FOUR CASES. ALBUMINURIA WAS USUALLY PRESENT TO A MILD DEGREE. THE WEIL-FELIX WAS FREQUENTLY OF LOW TITRE TO OXX WITH NO CO-AGGLUTININS. THERE WERE NO FATAL CASES. ALL THE PATIENTS WERE EXPOSED TO JUNGLE VECTORS DURING THEIR TRAINING. THESE CASES WOULD APPEAR TO BE A VERY MILD STRAIN OF TYPHUS-LIKE FEVER. THE SEROLOGICAL RESULTS ARE NOT CONCLUSIVE; THE TITRES ARE LOW, AND THOUGH SCRUB TYPHUS MIGHT BE CONSIDERED I AM NOT CONVINCED BY THESE RESULTS THAT THEY WERE CASES OF SCRUB TYPHUS AS SEEN ELSEWHERE.
SEN GUPTA (282) IN 1944 REPORTED A CASE OF TYPHUS FEVER COMPLICATING KALA AZAR. A CHILD AGED FOUR WITH KALA AZAR WAS GIVEN INJECTIONS OF AMINOSTIBUREA AND BECAME AFEBRIILE. AFTER ABOUT SEVEN WEEKS THE CHILD DEVELOPED FEVER AND A RASH ON THE FIFTH DAY WHICH GRADUALLY DISAPPEARED. WHAT SEEMED TO BE A SIGNIFICANT TITRE FOR OXK WAS FOUND ON THE SEVENTH DAY OF THE ILLNESS (1/400) WHICH FELL TO 1/25 FOUR DAYS LATER. IN VIEW OF THE DOUBTFUL RESULTS IN TWO CASES OF KALA AZAR WHICH I HAVE SEEN I AM INCLINED TO REGARD THIS CASE WITH SUSPICION THOUGH I AGREE THAT IT IS SUGGESTIVE CLINICALLY.

A CONFERENCE (258) HELD BY THE ARMY AUTHORITIES IN CEYLON IN FEBRUARY 1944 DISCLOSED THAT THERE HAD BEEN AN OUTBREAK OF SCRUB TYPHUS AMONGST EAST AFRICAN AND BRITISH TROOPS IN CEYLON DURING THE PREVIOUS MONTH. 713 CASES WERE SEEN AMONGST EAST AFRICANS AND 43 AMONGST BRITISH TROOPS. ESCHARS WERE SEEN IN 85% OF A SERIES OF 200 CASES, LYMPHADENITIS IN 80%, AND A RASH IN 6%. IN ANOTHER SERIES OF 430 CASES THE DESCRIPTIONS WERE SAID TO BE SIMILAR, BUT SOME CASES SHOWED SIGNS OF MENINGEAL IRRITATION. IN THESE CASES WITH MENINGEAL IRRITATION THE CEREBRO-SPINAL FLUID PRESSURE WAS RAISED. IN 7 CASES THE CEREBRO-SPINAL FLUID CELL COUNT
WAS 80 PER C.MM. OR OVER. A RASH WAS SEEN IN 7 CASES. ALBUMINURIA WAS NOTED IN 20 CASES ON ADMISSION AND RENAL FAILURE SYMPTOMS IN TWO CASES. THE OVERALL MORTALITY WAS REMARKABLY LOW, BEING ONLY 1.3%.

JACKSON (127) REPORTED IN 1945, 213 CASES SEEN IN GILGIT IN KASHMIR. THE MAXIMUM SEASONAL INCIDENCE WAS IN MARCH, APRIL, MAY AND JUNE. THE MORTALITY WAS LOW. THE DESCRIPTION OF THE DELIRIUM, THE RASH ON THE TRUNK ON THE FIFTH TO THE SEVENTH DAY, THE COMPLICATIONS, (PNEUMONIA AND SUPPURATIVE PAROTITIS) ARE CONSISTENT WITH THE OPINION OF JACKSON THAT IT WAS A LOUSE-BOURNE EXANTHEMATIC TYPHUS, IN SPITE OF THE LOW MORTALITY.

NOOR HUSSAIN (126) IN 1945 RECORDED THE EPIDEMIC IN KASHMIR IN 1942-43. THE CASES MAINLY OCCURRED IN WINTER. THERE WAS A MORTALITY OF 25% - 50%. THE LOCAL "GUJJARS" (NATIVES) WERE LOUSE INFESTED. MOST OF THE WEIL-FELIX RESULTS WERE POSITIVE FOR OX19 THOUGH A FEW WERE POSITIVE FOR OXK WITH CO-AGGLUTININS FOR OX2. HUSSAIN STATES THAT SAVOOR STUDIED THE KASHMIR TYPHUS IN 1944. THE NEIL-MOOSER TEST WAS NEGATIVE. HE STATES THAT 946 CASES WERE REPORTED WITH 320 DEATHS. THERE SEEMS LITTLE DOUBT THAT MANY OF THE
CASES WERE LIKELY TO BE LOUSE-BORNE EXANTHEMATIC TYPHUS, BUT THE CROSS AGGLUTINATION IN SOME CASES SUGGESTS THAT THESE WERE OF THE INDETERMINATE GROUP.

TATTERSALL AND PARRY (309) REPORTED IN 1945, AN OUTBREAK OF ONE HUNDRED AND TWENTY-ONE CASES IN A BRITISH REGIMENT "GEOGRAPHICALLY ADJACENT TO BURMA". THIS WAS NEAR IMPHAL IN MANIPUR STATE. THE FIRST CASE OCCURRED NINE DAYS AFTER THE UNIT ARRIVED FROM A "NON ENDEMIC AREA". THE LAST OCCURRED SEVENTEEN DAYS AFTER LEAVING.

THE CASES WERE SEEN DURING NOVEMBER AND DECEMBER AND WERE MAXIMUM TWELVE TO FIFTEEN DAYS AFTER ARRIVAL. IT IS OF CONSIDERABLE INTEREST TO HAVE A REPORT ON A LARGE NUMBER OF BRITISH CASES. A PRIMARY LESION WITH ESCHAR WAS SEEN IN 9% OF CASES. THE CLINICAL DESCRIPTION FOLLOWS IN GENERAL THE DESCRIPTIONS OF TYPHUS ELSEWHERE. THE ONSET WAS SUDDEN, HEADACHE WAS ALWAYS COMPLAINED OF AND PHOTOPHOBIA FREQUENTLY. A SORE THROAT WAS VERY COMMON, ADENITIS WAS ALMOST INVARIABLY PRESENT AND ALWAYS THERE WAS THE CHARACTERISTIC MENTAL APATHY. THE RASH APPEARED AS EARLY AS THE THIRD DAY AND PERSISTED FOR ELEVEN DAYS ON AN AVERAGE. IF SCANTY IT WAS CONFINED TO THE TRUNK, OTHERWISE IT WAS SEEN ALL OVER THE BODY.
IT WAS MAINLY MACULAR OR MACULO-PAPULAR. A FEW CASES WERE PURPURIC WITH PERSISTENT STAINING. THERE SEEMED TO BE NO RELATIONSHIP BETWEEN THE SEVERITY OF THE SYMPTOMS AND THE INTENSITY OF THE RASH. CHEST SIGNS WERE NOTED IN 88% OF CASES. NERVE DEAFNESS WAS NOTED IN 33% OF CASES. IN NO CASE WAS THE DEAFNESS PERMANENT. THE WEIL-FELIX WAS DIAGNOSTIC ON THE THIRTEENTH DAY. THERE WAS USUALLY A HIGH TITRE TO OXK, WITH NO CO-AGGLUTININS. THIS SUGGESTS A PURE SCRUB TYPHUS EPIDEMIC, WITH WHICH THE LOCUS, CONDITIONS AND CLINICAL APPEARANCES ARE IN AGREEMENT. THE WHITE BLOOD COUNTS WERE WITHIN NORMAL LIMITS. AN EOSINOPHILIA WAS RECORDED IN MANY CASES DURING CONVALESCENCE. THERE WERE ELEVEN DEATHS (9.9%). COMPLICATIONS WERE MINOR AND FEW, EXCEPT FOR A RETROBULBAR NEURITIS SEEN IN ONE CASE.

NAKED EYE AUTOPSY FINDINGS WERE OF CONGESTED LUNGS, DILATED RIGHT HEART, PETECHIAL HAEMORRHAGES IN THE STOMACH, ILEUM, CAECUM AND COLON, AND IN SEVERAL CASES, ENLARGED MESENTERIC GLANDS AND CONGESTION OF THE BRAIN AND MENINGES. SAVOOR (273) WAS ABLE TO DEMONSTRATE RICKETTSIAE FROM THE PERITONEAL EXUDATE OF MICE INFECTED FROM A CASE. A STRAIN
OF RICKETTSIA WAS ISOLATED IN THE AREA BY PARKER AND SAVOOR (239).

TATTERSALL (308) LATER REPORTED ONE THOUSAND CASES FROM THE AREA INCLUDING THOSE IN THE FIRST REPORT BUT INCLUDING INDIAN TROOPS. BETWEEN THE TWO REPORTS THERE WERE ONLY A FEW SIGNIFICANT DIFFERENCES. A RASH WAS SEEN IN 64% OF THE EUROPEAN CASES AND ONLY 31% OF THE INDIAN CASES. MORTALITY WAS 7% IN EUROPEAN CASES, AND 5% IN INDIAN CASES. IT IS SIGNIFICANT I THINK THAT THE WEIL-FELIX RESULTS SHOWED AS A RULE HIGH OXK AGGLUTININS AND NO CO-AGGLUTININS. IN SUCH A LARGE NUMBER OF CASES THE ABSENCE OF CO-AGGLUTININS IS SUGGESTIVE OF A PURE INFECTION OF SCRUB TYPHUS (OR TSUTSUGAMUSHI DISEASE). THERE IS NO DOUBT THAT THESE WERE CASES OF SCRUB TYPHUS, ALMOST CERTAINLY THE MITE WAS THE VECTOR, AND IT SEEMS LIKELY THAT THE EPIDEMIC WAS DUE TO CAMPING AND TRAINING CONDITIONS OF JUNGLE WARFARE DISTURBING THE NATURAL VECTOR HOST RELATIONSHIP.

KLEIN (138) DESCRIBED IN 1945 FORTY-ONE BRITISH CASES OUT OF TWO HUNDRED AND FIFTY-FIVE SEEN IN THE CHIN HILLS IN BURMA. CLINICALLY THEY RESEMBLED TATTERSALL AND PARRY'S (309) DESCRIPTION. KLEIN ALSO NOTED THE DIFFERENCE IN THE MORTALITY RATE
OF 12% FOR THE BRITISH CASES AS AGAINST 3.8% IN 184 INDIAN CASES. KLEIN SUSPECTED THAT THE "DREAD OF THE DISEASE" BY THE BRITISH CONTRIBUTED TO THIS HIGH FATALITY RATE. A SIMILAR "DREAD" AMONGST AMERICAN TROOPS IN THE SOUTH WEST PACIFIC WAS MENTIONED BY MAJOR GENERAL THOMPSON IN HIS CLOSING REMARKS AT THE CEYLON CONFERENCE (258) ON TYPHUS. THE MORBID ANATOMY IN KLEIN'S CASES WAS SIMILAR TO TATTERSALL AND PARRY'S CASES. MICROSCOPICALLY A MONONUCLEAR INFILTRATION WAS FOUND IN THE MUSCLE FIBRES OF THE HEART. IN TREATMENT PENICILLIN WAS TRIED, WITH NO EFFECT. THE EFFECT OF LUMBAR PUNCTURE WAS OFTEN DRAMATIC IN THOSE WITH A RAISED PRESSURE. THE IMPORTANCE OF A HIGH FLUID INTAKE WAS RECOGNISED.


MY OWN SERIES OF CASES IN CALCUTTA WAS DESCRIBED IN 1945 (LUSK) (165). THEY CONSISTED OF CASES OCCURRING IN A JUNGLE TRAINING AREA, EIGHTY MILES FROM CALCUTTA, TOGETHER WITH CASES OCCURRING IN CALCUTTA ITSELF. SEROLOGICALLY THE WEIL-FELIX RESULTS WERE USUALLY OXK ALONE, WITH IN SOME CASES CROSS AGGLUTINATION AND IN A FEW CASES AGGLUTINATION OF OX19 ONLY. THESE LATTER CASES WERE ON THE WHOLE MILDERTHAN THE AVERAGE AND WERE PROBABLY CASES OF THE MINDER MURINE TYPHUS. IT IS OF INTEREST HOWEVER, THAT CASES APPARENTLY OF SCRUB TYPHUS WERE FOUND IN GREATER CALCUTTA. THE MORTALITY OF ALL CASES WAS NEARLY FIFTEEN PER CENT. A RASH, SOMETIMES JUST VISIBLE, WAS SEEN IN ONLY THIRTEEN PER CENT OF THE CASES. IN ONE CASE WHICH AGGLUTINATED OX2 IN HIGH TITRE, A MARKED RASH WAS SEEN. THE CLINICAL DIFFERENCES BETWEEN THE SEROLOGICAL GROUPS ARE NOTED
LATER. A STRAIN OF RICKETTSSIA WAS ISOLATED BY PARKER (239) WITH SAVOOR'S ASSISTANCE FROM A CASE OCCURRING IN CALCUTTA.

ROY (268) IN 1946 SUMMARIZED THE INCIDENCE OF THE FEVERS OF THE TYPHUS GROUP IN INDIA. HE CONSIDERED THAT ALL INDIAN CASES REPORTED UP TO 1943 WERE LOUSE-BORNE. THIS IS A SOMEWHAT PECULIAR OPINION, UNSUPPORTED BY FACTS.

LOW (163) IN 1946 REPORTED TEN CASES AMONGST CIVILIANS IN CALCUTTA. HE MENTIONS AN UNPUBLISHED OUTBREAK AMONGST GREEK SAILORS OCCURRING IN 1942, AND THE BRITISH AND INDIAN MILITARY CASES IN 1942-43 AND TWO CASES DESCRIBED BY ROY IN 1944. SIX OF THESE CASES OF LOW'S OCCURRED IN THE CENTRE OF CALCUTTA. SEROLOGICALLY FIVE WERE OX19 ONE WITH CO-AGGLUTININS FOR OX2, ONE WAS OX2, AND FOUR WERE OXK. A RASH WAS SEEN IN TWO CASES. THERE WAS ONE DEATH. LUNGS SIGNS WERE COMMONLY PRESENT. THE RAT AND THE RAT FLEAS WOULD SEEM TO BE THE LIKELIEST HOST AND VECTOR TO SUSPECT, IN SOME OF THE CASES, BUT NO ATTEMPT HAS BEEN MADE TO TRACK DOWN THE VECTOR OR HOST. THE OXK CASES MAY HAVE BEEN SCRUB TYPHUS.

HEILIG (116) IN 1946 REPORTED A CASE FROM JAIPUR IN RAJPUTANA. HE
CONSIDERED THAT THE CASE RESEMBLED THOSE SEEN BY HIMSELF AND NAIDU IN MYSORE. THERE WAS A SIGNIFICANT WEIL-FELIX TITRE FOR OX19 ON THE ELEVENTH DAY FALLING TO NORMAL ON THE TWENTY-FIFTH DAY. HEILIG WAS UNABLE TO OBTAIN ANY RATS WHICH WERE SAID TO BE PRESENT IN THE HOME. THE PATIENT HAD CATTLE IN HIS HOUSE AND TWENTY-FOUR TICKS WERE TAKEN FOR INOCULATION EXPERIMENTS AT THE HAFFKINE INSTITUTE. RESULTS WERE INCONCLUSIVE. THE RAT AND RAT FLEA WOULD SEEM TO BE THE POSSIBLE HOST AND VECTOR.

MACKIE (169) AND HIS COLLABORATORS OF THE UNITED STATES OF AMERICA TYPHUS COMMISSION, INDIA BURMA THEATRE, IN 1946 PUBLISHED A PRELIMINARY REPORT ON THEIR INVESTIGATIONS. THE SERIOUSNESS OF THIS DISEASE IN THE JUNGLE FIGHTING ON THE BURMA FRONT HAD BEEN RECOGNISED AND A COMPREHENSIVE INVESTIGATION WAS CARRIED OUT BY A LARGE TEAM. THEY FOUND THAT SCRUB TYPHUS WAS CONTRACTED IN EVERY MONTH OF THE YEAR. THE PEAK TIMES OF INFECTION WERE HOWEVER AT THE BEGINING AND END OF THE MONSOON, AT THE TIME WHEN TROMBICULA DELIENSIS WALCH WAS FOUND IN GREATEST ABUNDANCE.

THE CASE AGAINST THE LARVAL T.
DELIENSIS AS THE VECTOR WAS PROVED UP TO THE HILT. TRANSOVARIAL TRANSMISSION WAS PROVED. TWO MAMMALS, RATTUS FLAVIPECTUS YUNNANENSIS (ANDERSON), (THE YUNNAN BUFF-BREASTED RAT,) AND TUPAIA BELANGERI VERSURAE (THOMAS), (THE ASSAMESE TREE SHREW,) WERE THE ONLY ONES FOUND TO BE NATURALLY INFECTED WITH RICKETTSIA ORIENTALIS. MACKIE AND HIS COLLABORATORS CONSIDERED THAT THE DENSITY OF THE TROMBICULA DELIENSIS WALCH POPULATION IN ANY AREA MIGHT PROVIDE AN APPROXIMATE INDEX OF THE RISK OF INFECTION. THEY WERE ABLE TO SHOW THAT IN NORTHERN BURMA SCRUB TYPHUS WAS ENDEMIC. SAYERS (276) IN THE DISCUSSION ON THE PAPER READ BY MACKIE AT A MEETING OF THE ROYAL SOCIETY OF TROPICAL MEDICINE WAS ABLE TO STATE THAT THREE HUNDRED MILES FURTHER SOUTH OF THE SITE OF THE AMERICAN TEAMS WORK WAS ANOTHER ENDEMIC AREA. FELIX (88) IN THE SAME DISCUSSION COMMENTING ON THE DIFFERENT 'SEASONAL' INCIDENCE BETWEEN THE AMERICAN AND BRITISH ARMIES, CONSIDERED THAT THE TWO SETS OF FIGURES WERE PERHAPS NOT IN FACT STRICTLY COMPARABLE.

SEE TABLE THIRTEEN.

ALSO SEE MAPS SEVEN, EIGHT AND NINE.
<table>
<thead>
<tr>
<th>SUGGESTED VECTOR IN CASES REPORTED IN THE INDIAN LITERATURE 1932-1946.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BULBIR SINGH</strong></td>
</tr>
<tr>
<td><strong>JACKSON</strong></td>
</tr>
<tr>
<td><strong>NOOR HUSSAIN</strong></td>
</tr>
<tr>
<td><strong>BOYD</strong></td>
</tr>
<tr>
<td><strong>STOTT</strong></td>
</tr>
<tr>
<td><strong>PATEL</strong></td>
</tr>
<tr>
<td><strong>SHARMA</strong></td>
</tr>
<tr>
<td><strong>WOODHEAD AND DUTT</strong></td>
</tr>
<tr>
<td><strong>MAITRA AND SEN GUPTA</strong></td>
</tr>
<tr>
<td><strong>SONI</strong></td>
</tr>
<tr>
<td><strong>LUSH</strong></td>
</tr>
<tr>
<td><strong>HEILIG</strong></td>
</tr>
<tr>
<td><strong>LOW</strong></td>
</tr>
<tr>
<td><strong>BOYD</strong></td>
</tr>
<tr>
<td><strong>CHRISTIAN</strong></td>
</tr>
<tr>
<td><strong>MARTIN AND ANDERSON</strong></td>
</tr>
<tr>
<td><strong>WILSON</strong></td>
</tr>
<tr>
<td><strong>BUSI</strong></td>
</tr>
<tr>
<td><strong>MACNAMARA</strong></td>
</tr>
<tr>
<td><strong>PANDALAI</strong></td>
</tr>
<tr>
<td><strong>MAITRA AND SEN GUPTA</strong></td>
</tr>
<tr>
<td><strong>KAPILA AND MAITRA</strong></td>
</tr>
<tr>
<td><strong>WEBSTER</strong></td>
</tr>
<tr>
<td><strong>SHARMA</strong></td>
</tr>
<tr>
<td><strong>WOODHEAD AND DUTT</strong></td>
</tr>
<tr>
<td><strong>GURBUKSH SINGH</strong></td>
</tr>
<tr>
<td><strong>BARO</strong></td>
</tr>
<tr>
<td><strong>SEN GUPTA</strong></td>
</tr>
<tr>
<td><strong>OUTBREAK IN ARMY IN CEYLON</strong></td>
</tr>
<tr>
<td><strong>TATTERSAL AND PARry</strong></td>
</tr>
<tr>
<td><strong>TATTERSAL</strong></td>
</tr>
<tr>
<td><strong>KLEIN</strong></td>
</tr>
<tr>
<td><strong>DESMUKH</strong></td>
</tr>
<tr>
<td><strong>LUSH</strong></td>
</tr>
<tr>
<td><strong>LOW</strong></td>
</tr>
</tbody>
</table>

**MITER PROVED VECTOR.**

**MACKIE AND COLLABORATORS** | ASSAM, BURMA.

**VECTOR UNKNOWN, BY ANECDOTAL TEST POSSIBLE IN SOME CASES MITE NOT ABSOLUTELY IMPOSSIBLE.**

| **BOYD** | POONA, AHMEDNAGAR, S. INDIA |
| **CHRISTIAN** | DELHI, FIVE CASES, BAREILLY |
| **BUSI** | SIMLA HILLS |
| **BLEWITT** | AHMEDNAGAR |
| **BEVERIDGE AND UNDERHILL** | CENTRAL INDIA |
| **SARKAR** | RAIPUR CENTRAL PROVINCES |
| **WOODHEAD AND DUTT** | ASSAM |
| **MAITRA AND SEN GUPTA** | BURMA |
| **GURBUKSH SINGH** | BURMA |
| **SONI** | ASSAM |
| **HUSSAIN** | BURMA |
| **LUSH** | CALCUTTA |
| **LOW** | CALCUTTA |
| **CURRAN** | KASHMIR |
| **HEILIG AND NAIDU** | JABALPORE |
| **YACOBI** | MUZAFFARGARH PUNJAB. |

**INSUFFICIENT INFORMATION TO ENABLE ANY OPINION TO BE FORMED AS TO THE POSSIBLE VECTOR.**
MAP SHOWING LOCATION OF CASES REPORTED AFTER STANDARDIZATION OF WEIL-FELIX REACTION IN INDIA, WHERE A FLEA VECTOR SEEMS LIKELY OR POSSIBLE.
MAP SHOWING LOCATION OF CASES REPORTED AFTER STANDARDIZATION OF WEIL-FELIX REACTION IN INDIA, WHERE A MITE VECTOR IS PROBABLE. IN AREA OF MACKIE'S INVESTIGATION THE MITE PROVED TO BE THE VECTOR.
Map showing location of cases reported after standardization of Weil-Felix reaction in India, where the vector is uncertain. These cases are mainly of the indeterminate serological group. A tick is conjectured but so far no proof has been obtained.
FROM THIS SUMMARY OF THE INDIAN LITERATURE IT IS SEEN THAT SCRB TYPHUS IS PRESENT IN THE JUNGLES OF ASSAM AND BURMA AND ITS VECTOR AND HOST HAVE BEEN ESTABLISHED. STRAINS FROM SEROLOGICAL OXK CASES HAVE BEEN ISOLATED IN CALCUTTA AND SIMLA. IT IS PROBABLE THAT THE VECTOR IN THESE AREAS, THOUGH PROOF IS STILL LACKING, IS THE LARVAL MITE. FROM SEROLOGICAL RESULTS SCRUB TYPHUS IS PRESENT IN OTHER PARTS OF INDIA AND CEYLON. A MURINE TYPHUS STRAIN HAS BEEN ISOLATED FROM RATS AND RAT FLEAS IN SIMLA. ON CLINICAL AND SEROLOGICAL GROUNDS MURINE TYPHUS IS CONSIDERED TO OCCUR IN VARIOUS PARTS OF INDIA AND CEYLON.

ONE IS CONSCIOUS HOWEVER THAT A GREAT MANY OF THE CASES REPORTED IN THE INDIAN LITERATURE ARE OF THE INDETERMINATE SEROLOGICAL TYPE OF FELIX'S CLASSIFICATION, (LOW TITRES WITH ONE OR ALL OF THE ORDINARY PROTEUS SUSPENSIONS, OR HIGH MIXED TITRES.) ONE ASSUMES THAT THESE CONTRACT THE DISEASE FROM A VECTOR OTHER THAN THE LOUSE, RAT FLEA OR LARVAL MITE. WHETHER A TICK OR TICKS PLAY A PART IS UNKNOWN.

THE DESCRIPTION OF PERSONAL CASES SEEN IN CALCUTTA DURING THE WAR OF 1939-46

WHILE IN CHARGE OF A MEDICAL DIVISION OF
AN INDIAN MILITARY HOSPITAL IN CALCUTTA IN 1943-44, SIXTY CASES WERE FORWARDED FROM A FIELD AMBULANCE STATIONED EIGHTY MILES TO THE NORTH, NEAR A VILLAGE CALLED JESSORE.

SEE MAP TEN.

A BRIGADE, CARRYING OUT JUNGLE TRAINING EXERCISES, WAS STATIONED AT GINGERGATCHA GHAT, NEAR JESSORE. 'GHAT' MEANS FERRY OR BATHING PLACE AND INDICATES AS A RULE THAT THE PLACE IS NEAR A RIVER OF SOME SIZE. FIFTY-FOUR FURTHER CASES WERE SEEN FROM GREATER CALCUTTA ITSELF. THESE ONE HUNDRED AND FOURTEEN CASES WERE THE SUBJECT OF A ROUTINE REPORT TO THE ARMY AUTHORITIES, WHO AT A LATER DATE SUBMITTED IT FOR PUBLICATION TO THE INDIAN MEDICAL GAZETTE. IN THAT REPORT AN ATTEMPT WAS MADE TO DESCRIBE THE DISEASE AS SEEN IN THAT AREA, WITHOUT ATTEMPTING TO DIFFERENTIATE BETWEEN THE SEROLOGICAL GROUPS. A NOTE OF THE PROMINENT MORBID ANATOMICAL FINDINGS WAS ADDED. IN THIS THESIS AN AMPLIFICATION OF THE PATHOLOGICAL FINDINGS WILL BE MADE, BY THE STUDY OF THE MORBID HISTOLOGY OF THE SECTIONS FROM THREE FATAL CASES.

NATIONALITY OF PATIENTS.

ALL THE PATIENTS WERE NATIVES OF INDIA WITH FIVE EXCEPTIONS, ONE GURKAH FROM NEPAL, TWO NATIVES OF BURMA AND TWO NATIVES OF WEST
MAP TEN

MAP SHOWING LOCATION OF CASES SEEN BY MYSELF WHILE STATIONED IN CALCUTTA IN 1943-1944.

LOCATION IN INDIA OF MAP SHOWN ABOVE
THE WEIL-FELIX RESULTS.

The majority of cases agglutinated OXK in diagnostic titre. Seventy cases (approximately sixty-two percent) agglutinated OXK alone, and in a further twenty-six cases (twenty-two percent) there was co-agglutination of OX19 and occasionally OX2. Ten cases (approximately nine percent) agglutinated OX19 alone. In two cases predominantly OX19 there were co-agglutinins for OXK present. In one case there was agglutination of OX2 with co-agglutination of OX19. Five cases were diagnosed on clinical and autopsy findings.

See Table Fourteen.

In some cases the titres were high; estimations however were never carried out beyond 1/5000. Titres were considered to be low when the highest reading was 1/500. A titre of 1/1000 or over was considered to be a high titre. It was noted that all the serologically proved fatal cases were, with one exception, OXK with no cross agglutination. The exception was a case which was clinically distinct, and where OX2 rose to 1/1000. Co-agglutination of OX19 was noted in this case on one occasion to 1/125. A table shows the number of cases and their degree of severity in the various serological groups.
### Table Fourteen

SEROLOGICAL RESULTS IN ONE HUNDRED AND FOURTEEN CASES SEEN IN CALCUTTA.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>OXK Alone</td>
<td>70 (62%)</td>
</tr>
<tr>
<td>OXK with co-agglutination of OX2 and OX19</td>
<td>26 (22%)</td>
</tr>
<tr>
<td>OX19 Alone</td>
<td>10 (9%)</td>
</tr>
<tr>
<td>OX19 with co-agglutination of OXK</td>
<td>2 (1.7%)</td>
</tr>
<tr>
<td>OX2 with co-agglutination of OX19</td>
<td>1 (0.9%)</td>
</tr>
<tr>
<td>Clinical Cases</td>
<td>5 (4.4%)</td>
</tr>
</tbody>
</table>

### Table Fifteen

THE NUMBER OF CASES AND DEGREE OF SEVERITY IN THE VARIOUS SEROLOGICAL GROUPS

<table>
<thead>
<tr>
<th>Condition</th>
<th>Mild Cases</th>
<th>Moderate Cases</th>
<th>Severe Cases</th>
<th>Very Severe Cases</th>
<th>Fatal Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>OXK High Titre</td>
<td>3</td>
<td>16</td>
<td>1</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>OXK Low Titre</td>
<td>4</td>
<td>30</td>
<td>2</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>OXK with cross agglutinins High Titre</td>
<td>13</td>
<td>2</td>
<td>1</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td>OXK with cross agglutinins Low Titre</td>
<td>7</td>
<td>3</td>
<td>1</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>OX19 High Titre</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>OX19 Low Titre</td>
<td>2</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>OX2 High Titre</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>OX2 with low titre and cross agglutination of OX19</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Clinical Cases</td>
<td>5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
SEE TABLE FIFTEEN.

It was noted that the highest titres in the Weil-Felix test were often seen towards the end of the second week. Considerable variation was of course seen.

One very interesting case was seen sent in from Jessore. On the sixth day Ox19 and Oxk were each agglutinated to 1/125. On the ninth day Ox2 was agglutinated to 1/250, Ox19 to 1/125 and Oxk to 1/50 only. Thereafter titres to all suspensions were low until the fortieth day when Oxk rose again to 1/125 and then to 1/500 on the sixty-second day, and 1/1000 on the sixty-sixth day.

SEE TABLE SIXTEEN.

Discussion of the possible vector in the cases seen.

No positive information leading one to suspect a vector was obtained. Nor were any ectoparasites seen, not lesions to suggest the bite of any of the recognised vectors, lice, fleas, ticks or mites. This lack of positive information is common in the Indian literature prior to the outbreaks seen in the Burma campaign by Gurbuksh Singh (109), Klein (138), Tattersall (308) Tattersall and Parry (309) and Deshmukh (69). In their reports they all record eschars and local
LESIONS. THE REPORTS FROM INDIA DURING THE WAR YEARS OF BARDAN (16) AND LOW (163) REPEAT THE PEACE TIME FINDINGS OF NO LOCAL LESION. THIS IS NOT ENTIRELY EXPLAINED BY THE DIFFERENT LIVING CONDITIONS OF THE PATIENTS. TATTERSALL'S, GURBUKSH SINGH'S, KLEIN'S AND DESHMUKH'S WERE EXISTING UNDER WARTIME JUNGLE CONDITIONS. BARDAN'S, LOW'S AND SOME OF MY OWN CASES WERE LIVING IN BARRACKS, QUARTERS, OR THEIR HOMES. SIXTY OF MY CASES HOWEVER, WERE FROM A JUNGLE TRAINING CAMP AND MOST OF THEM WERE CARRYING OUT EXERCISES UNDER CONDITIONS SIMULATING JUNGLE WARFARE. THERE IS NO OBVIOUS EXPLANATION OF THIS DISCREPANCY. WE MAY ASSUME AS POSSIBLE A VARIATION IN THE VECTOR, OR ITS MORE FAVOURABLE OPPORTUNITY UNDER CONDITIONS OF ACTUAL WARFARE FOR A MITE TO BITE MORE EFFECTIVELY. EITHER OF THESE SUGGESTIONS AWAITS FURTHER INVESTIGATION.

THE ISOLATION OF A STRAIN OF RICKETTSIA FROM ONE OF MY CASES BY PARKER AND SAVOOR (239) IS REPORTED ON BY SMADEL, RIGHTS AND JACKSON (288). THEY FOUND THE STRAIN INDISTINGUISHABLE FROM AN ASSAM STRAIN OF TATTERSALL AND PARRY ON THE BASIS OF COMPLEMENT FIXATION AND CROSS IMMUNITY TESTS. IT WOULD BE PECULIAR IF A TYPICAL SCRUB TYPHUS STRAIN WERE TO BE ISOLATED FROM A CASE IN WHICH THE MITE WAS NOT THE VECTOR.
DISCUSSION OF THE POSSIBLE HOST OF THE VECTOR.

The only suggestive evidence of a possible host in my cases in Greater Calcutta, (as distinct from the Jessore cases), is the finding that the largest number of cases (seven) from one unit, were housed in quarters, which on their own statements were heavily infested with rats. I was to confirm this later in conversation with their commanding officer. Unfortunately it was impossible to carry out any investigations on these rats.

Goyal (105), as already mentioned, had reported on epizootic rickettsial infection in Calcutta wild rats which died out in 1940.

The serological results in these seven cases were of interest. One case agglutinated Proteus OX2 with cross agglutination for OX19. Two showed agglutination for OXK and cross-agglutination for OX19. One agglutinated OX19 only. Three agglutinated OXK only.

See Table Seventeen.

There is thus very little factual information on the mode of transmission and the vector in cases in Bengal. By analogy and on circumstantial evidence it is highly probable that the larval mite is a vector of the cases in contact with jungle which agglutinate Proteus OXK. When one refers to
**Table Seventeen**

SEROLOGICAL RESULTS IN SEVEN CASES IN ONE UNIT IN CALCUTTA WHO OCCUPIED QUARTERS SAID TO BE OVERRUN WITH RATS.

<table>
<thead>
<tr>
<th>Day of Disease</th>
<th>5</th>
<th>10</th>
<th>12</th>
<th>13</th>
<th>15</th>
<th>16</th>
<th>18</th>
<th>19</th>
<th>21</th>
<th>22</th>
<th>23</th>
<th>24</th>
<th>27</th>
</tr>
</thead>
<tbody>
<tr>
<td>OX2</td>
<td>1/25</td>
<td>1/25</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1/25</td>
</tr>
<tr>
<td>OX19</td>
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<td>OXK</td>
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<td>OXK</td>
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</tr>
</tbody>
</table>

DEATH

FROM BLOOD AT AUTOPSY
JUNGLE ONE MEANS LAND WHICH HAS BEEN UNDIS-
turbed. It is probable that murine typhus
occurs in Calcutta and Bengal. Other vectors
than the larval mite and rat flea may operate.
It is possible that as Felix (86) and Pijper
and Dau (247) suggest in the case of typhus
fevers of South Africa, modifications exist
when comparison is made with the groups of the
disease as seen elsewhere. Further investi-
gations are required before any firm conclu-
sions can be drawn about the vectors of typhus
like fevers in Calcutta and Bengal.

SEASONAL INCIDENCE.

The fortnightly incidence is shown in
Tables Eighteen and Nineteen. These tables
show that while in Jessore, most of the
(Fifty one out of sixty) cases occurred between
the beginning of August and the end of October.
In Calcutta cases were seen up to the end of
December. In both groups, cases with main
agglutinins for OKK with cross-agglutination
for OX2, and/or OX19 were seen almost entirely
in September or October. The scatter of the
cases in the Calcutta group would at first
suggest the possibility that the vector may not
be the larval mite of Tsutsugamushi disease; or
that the bionomics of the mite in Calcutta, if
it be the vector, are not strictly comparable
<table>
<thead>
<tr>
<th>MONTH</th>
<th>FORTNIGHTLY PERIODS</th>
<th>NUMBER OF OXK CASES</th>
<th>NUMBER OF OXK CASES WITH CROSS AGGLUTININS</th>
<th>NUMBER OF OX49 CASES</th>
<th>NUMBER OF CLINICAL CASES</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>JUNE</td>
<td>16 30</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
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<tr>
<td>JULY</td>
<td>16 15 31</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td></td>
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<tr>
<td>AUGUST</td>
<td>16 15 30</td>
<td>6</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>SEPTEMBER</td>
<td>16 15 31</td>
<td>3</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>OCTOBER</td>
<td>16 15 30</td>
<td>6</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>NOVEMBER</td>
<td>16 15 30</td>
<td>36</td>
<td>19</td>
<td>3</td>
<td>2</td>
<td>60</td>
</tr>
</tbody>
</table>

TABLE EIGHTEEN

FORTNIGHTLY INCIDENCE OF THE CASES FROM JESSORE.
<table>
<thead>
<tr>
<th>MONTH</th>
<th>FORTNIGHTLY PERIODS</th>
<th>NUMBER OF OXK CASES</th>
<th>NUMBER OF OXK CASES WITH CROSS AGGLUTININS</th>
<th>NUMBER OF OX19 CASES</th>
<th>NUMBER OF OX10 CASES WITH LOW CROSS AGGLUTININS</th>
<th>NUMBER OF CLINICAL CASES</th>
<th>TOTAL</th>
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<tbody>
<tr>
<td>JULY</td>
<td>1, 31</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>AUGUST</td>
<td>16, 15</td>
<td>3</td>
<td>2</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>SEPTEMBER</td>
<td>15, 1</td>
<td>2</td>
<td>6</td>
<td>3</td>
<td>3</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td>OCTOBER</td>
<td>1, 15, 30</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>NOVEMBER</td>
<td>1, 15, 31</td>
<td>4</td>
<td>1</td>
<td>1</td>
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<td>1</td>
<td>3</td>
</tr>
<tr>
<td>DECEMBER</td>
<td>1, 31</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
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</tbody>
</table>

**Table Nineteen**

Fortnightly Incidence of the Cases from Greater Calcutta.
EFFECTED. FOUR OF MY CASES IN CALCUTTA (SEROLOGICALLY OXK), TWO OF WHICH WERE FATAL, CAME FROM A UNIT, RECENTLY ARRIVED, WHO CLEARED SOME BAMBOO JUNGLE NEAR THEIR CAMP AT TOLLYGUNGE, A SUBURB OF CALCUTTA. THESE OBSERVATIONS SEEM CONSISTENT WITH A DISTURBANCE OF THE BIONOMICS OF SUCH A HOST VECTOR CYCLE AS THE RAT AND THE LARVAL MITE. FURTHER ENQUIRY IS NEEDED, BUT MEANTIME I FEEL THAT IT MUST NOT BE ASSUMED THAT THE CASE AGAINST THE LARVAL MITE AS THE VECTOR, HAS BEEN PROVED IN BENGAL, THOUGH BY ANALOGY IT IS HIGHLY SUGGESTIVE.

CLINICAL DESCRIPTION OF THE CASES.

THERE WAS NO OBVIOUS DIFFERENCE IN THE VARIOUS SEROLOGICAL TYPES SEEN. IT COULD PERHAPS BE SAID THAT THE CASES WHICH AGGLUTINATED PROTEUS OX19 ONLY, GAVE ONE THE IMPRESSION OF BEING LESS SEVERELY ILL. NONE OF THE TEN CASES HAVING PROTEUS OX19 AS THE SOLE OR MAIN AGGLUTININ DIED.

THE ONSET.

THE ONSET IS USUALLY SUDDEN (79.8%) AND IS NOT INFREQUENTLY ASSOCIATED WITH RIGORS (33.1/3%). IN FOUR CASES WITH A HISTORY OF ONSET WITH RIGORS, REPEATED EXAMINATIONS OF PERIPHERAL BLOOD FILMS WERE NEGATIVE FOR MALARIAL PARASITES. THIS SUGGESTS THAT RIGORS
MAY OCCUR DURING THE ONSET OF TYPHUS BUT MALARIA MAY BE CONSIDERED A LIKELY CAUSE OF RIGORS IN AN AREA WHERE MALARIA IS ENDEMIC. AT AN EARLY STAGE IN NEARLY ALL CASES SLOW CEREBRATION AND STUPOR WAS OBVIOUS TO A GREATER OR LESSER EXTENT, AS HAS BEEN NOTED BY ALL OBSERVERS OF THE DISEASE. THIS MAKES INFORMATION SOMETIMES DIFFICULT TO ELICIT, AND ONE GETS AN IMPRESSION THAT PAIN IS ONLY COMPLAINED OF IF SEVERE.

PAINFUL SYMPTOMS

HEADACHE IS OF COURSE OFTEN PRESENT WITH FEVER FROM ANY CAUSE. IT WAS NOTED IN 34% OF CASES. GENERALIZED PAIN IN THE BODY WAS COMPLAINED OF IN 13% OF CASES, DEEP SEATED PAIN IN THE CHEST IN 8% OF CASES, AND ABDOMINAL PAIN NOT PARTICULARLY LOCALIZED IN 7% OF CASES. ONE CASE COMPLAINED OF PAIN IN THE LIMBS AND JOINTS AND ONE CASE OF PAIN IN THE CALVES.

THE EYES AND THROAT.

CONJUNCTIVAL CONGESTION WAS SEEN IN 17% OF CASES AND PHARYNGEAL CONGESTION IN 8%.

THE TONGUE.

THE TONGUE WAS COATED IN 74% OF CASES.

THE RESPIRATORY TRACT.

SIGNS OF BRONCHITIS WERE A VERY CONSTANT FINDING AT SOME PERIOD DURING THE DISEASE. COUGH WAS PRESENT IN 22% OF CASES BUT WAS NOT USUALLY TROUBLESOME TO THE PATIENT. IT WAS
USUALLY UNPRODUCTIVE. IN ONE CASE THE SPUTUM WAS TINGED WITH BLOOD, AND IN ONE CASE IT WAS RUSTY. IN THE FIRST INSTANCE EXAMINATIONS OF THE SPUTUM AND AN X-RAY DID NOT SUGGEST TUBERCULOSIS, AND THE PATIENT RECOVERED. IN THE SECOND CASE PHYSICAL SIGNS WERE NOT DIAGNOSTIC OF SECONDARY PNEUMONIA, NOR WAS THE CLINICAL APPEARANCE SUGGESTIVE OF PNEUMONIA. IT IS POSSIBLE HOWEVER THAT THE RUSTY SPUTUM WAS DUE TO A SMALL UNRECOGNISED PATCH OF LOBAR PNEUMONIA. SIGNS OF BRONCHITIS, (MEDIUM AND HIGH PITCHED RHONCHI, AND NON RESONANT MEDIUM OR COARSE CREPITATIONS) WERE PRESENT IN VARYING DEGREE AND IN VARIOUS COMBINATIONS IN 62% OF CASES. THESE SIGNS MAY APPEAR DURING THE FIRST FEW DAYS OF THE DISEASE, BUT THEIR APPEARANCE MAY BE DELAYED FOR SEVEN TO TEN DAYS. DURING THE COURSE OF THE DISEASE SIGNS SUGGESTIVE OF CONGESTION OF THE LUNGS MAY DEVELOP IN A FEW CASES. SLIGHT IMPAIRMENT OF THE PERCUSSION NOTE WAS FOUND IN 16% OF CASES AND IN A FEW INSTANCES FINE CREPITATIONS COULD BE HEARD WHERE THE OTHER ACCOMPANIMENTS WERE NOT TOO LOUD. ALTERATION IN VOCAL FREMITUS AND VOCAL RESONANCE WAS NEVER OBVIOUS EXCEPT FOR A SLIGHT DIMINUTION IN SOME CASES. IN A FEW CASES WHERE THE LUNG SIGNS WERE UNILATERAL THIS WAS EASY TO ELICIT. IN CASES WITH BILATERAL LESIONS IT WAS NOT SO DISTINCTIVE.
SOMETIMES PNEUMONIA WAS SUSPECTED WHEN THE PERCUSSION NOTE BECAME IMPAIRED, AND SULPHAPYRIDINE WAS EXHIBITED, BUT NO APPARENT IMPROVEMENT WAS EVER SEEN IN THE PATIENT'S GENERAL CONDITION, OR ANY OBVIOUS CHANGE IN THE PHYSICAL SIGNS TOWARDS FRANK CONSOLIDATION. IN FIFTEEN OUT OF SIXTEEN AUTOPSIES NO NAKED EYE APPEARANCES SUGGESTIVE OF PNEUMONIA WERE SEEN. THE LUNGS WERE SECTIONED IN SEVERAL CASES. IN ONE CASE EARLY BRONCHO-PNEUMONIA WAS REPORTED. CLINICALLY THEREFORE PNEUMONIA WAS NOT A COMPLICATION IN MY CASES IN CALCUTTA.

COARSE PLEURAL FRICTION WAS AUDIBLE IN ONE CASE. IN FIVE CASES AT AUTOPSY RECENT FINE PLEURAL ADHESIONS WERE SEEN.

THE LYMPHATIC GLANDS.

SUPERFICIAL GLANDULAR ENLARGEMENT WAS CONSPICUOUSLY ABSENT. IN ONLY TWO CASES WAS THERE A MODERATE DEGREE OF ENLARGEMENT OF CERVICAL, AXILLARY AND INGUINAL GLANDS. IN ONE CASE THE LYMPH GLANDS IN THE LEFT GROIN AND LEFT AXILLA WERE ENLARGED AND IN ONE CASE THE CERVICAL GLANDS WERE ENLARGED WITH SLIGHT ENLARGEMENT OF THE AXILLARY GLANDS. THIS FINDING IS IN AGREEMENT WITH BARDAN'S (16) OBSERVATIONS FROM THE UNITED PROVINCES, AND LOW'S (163) CALCUTTA CASES, BUT IN CONTRARY TO THE FINDINGS OF TATTERSALL (308), 92% OF WHOSE CASES HAD GLANDULAR ENLARGEMENT.
KLEIN (138), ALL OF WhOSE CASES SHOWED ENLARGEMENT, DESMUKH (69) WHOSE CASES USUALLY HAD Lymphadenitis, and GURBukSH SinGh (109) 82% OF WhOSE CASES HAD GLANDULAR ENLARGEMENT. A DEGREE OF SUPERFICIAL GLANDULAR ENLARGEMENT IS COMMON IN NATIVES OF INDIA, AND IN JUDGING OF WHETHER THERE IS ENLARGEMENT IT IS ADVISABLE TO OCCASIONALLY EXAMINE SEVERAL NORMAL INDIVIDUALS AS CONTROLS.

THE RASH.

IN FIFTEEN CASES (13%) A RASH WAS NOTED. THIS CONTRASTS WITH TATTERSALL'S (308) SERIES, WHERE IN INDIAN TROOPS A RASH WAS SEEN IN 31% OF CASES, AND IN EUROPEAN TROOPS IN 64% OF CASES. KLEIN (138) SAW A RASH IN FORTY OUT OF FORTY-ONE CASES IN EUROPEANS IN BURMA. DESMUKH (69) REPORTED A RASH IN 7% OF 200 CASES SEEN IN EAST AFRICAN TROOPS ON THE BURMA FRONT. BARDAN (16) DID NOT SEE A RASH IN FORTY-ONE CASES IN THE UNITED PROVINCES.

SKIN, WHICH DISAPPEARED ON PRESSURE, AND SLOWLY REAPPEARED. SUPERIMPOSED ON THE ERYTHEMA FLAT PAPULES WERE SEEN ON THE TRUNK AND EXTREMITIES. THEY WERE BROWN IN COLOUR AND IN DIAMETER WERE ABOUT THE SIZE OF A SIXPENCE. THE RASH WAS FADING IN INTENSITY BUT WAS STILL DISTINCT WHEN HE DIED ON THE 21ST DAY OF THE DISEASE.

SEE SKETCH ONE.

THE RASH IN THE OTHER CASES WERE VERY MUCH LESS DISTINCT. IN A FEW CASES IT WAS ONLY TO BE SEEN IF LOOKED FOR WITH CARE. SEVEN OF THESE CASES WERE SEROLOGICALLY OXK. FOUR CASES HAD OXK AS A MAIN AGGLUTININ WITH OX19 AS A CO-AGGLUTININ. TWO CASES HAD OXK AS A MAIN AGGLUTININ WITH CO-AGGLUTININS FOR OX2 AND OX19. IN ONE CASE THE DIAGNOSIS WAS MADE ON CLINICAL AND AUTOPSY FINDINGS.

SEE TABLE TWENTY.

THE DAY OF ONSET OF THE RASH COULD NOT BE DETERMINED WITH ANY DEGREE OF ACCURACY. THIS WAS PARTLY DUE TO THE DIFFICULTY IN SEEING THE RASH ON A PIGMENTED SKIN. IT WAS NOTED IN NINE CASES, ON ADMISSION FROM A FIELD AMBULANCE. THE RASH WAS SOMETIMES EXTREMELY INDEFINITE. IN TWO CASES WHERE THE DAY OF ONSET COULD BE FIXED IT WAS SEEN ON THE SEVENTH AND EIGHTH DAY RESPECTIVELY. IT CONSISTED OF NUMEROUS FAINT MACULES USUALLY CIRCULAR, OCCASIONALLY OVAL, OF 0.2 TO 0.3 CM. IN DIAMETER. THESE WERE
Impression of the rash seen in the case which agglutinated OX2 in high titre. There was a degree of erythema with superimposed papules in a relatively fair skinned Oriental.
TABLE TWENTY
WEIL-FELIX RESULTS IN THE FIFTEEN CASES WITH
A RASH.

<table>
<thead>
<tr>
<th>WEIL-FELIX RESULTS</th>
<th>NUMBER OF CASES</th>
</tr>
</thead>
<tbody>
<tr>
<td>OXK</td>
<td>7</td>
</tr>
<tr>
<td>OX2 with low co-agglutinins for OX19</td>
<td>1</td>
</tr>
<tr>
<td>OXK with co-agglutinins for OX19</td>
<td>4</td>
</tr>
<tr>
<td>OXK with co-agglutinins for OX2 and OX19</td>
<td>2</td>
</tr>
<tr>
<td>CLINICAL CASES</td>
<td>1</td>
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</tbody>
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TABLE TWENTY ONE
TABLE SHOWING DEGREE OF SPLENIC ENLARGEMENT
SEEN AT AUTOPSY IN SIXTEEN CASES.
(1) CASES WITH CO-INCIENT MALARIAL PARAZITAEMIA.
(2) CASES OF TYPHUS SUSPECTED TO HAVE COINCIDENT
MALARIA.
(3) CASES OF TYPHUS WHERE MALARIA WAS EXCLUDED.
(4) CASES DIAGNOSED AS TYPHUS ON CLINICAL AND
AUTOPSY FINDINGS IN WHICH ACTIVE MALARIA WAS
EXCLUDED.

<table>
<thead>
<tr>
<th>SPLEEN ( \downarrow )</th>
<th>(1) WITH MALARIAL PARAZITAEMIA</th>
<th>(2) CO-INCIENT MALARIA SUSPECTED</th>
<th>(3) MALARIA EXCLUDED</th>
<th>(4) CLINICAL TYPHUS ( \text{ACTIVE MALARIA EXCLUDED} )</th>
</tr>
</thead>
<tbody>
<tr>
<td>X5</td>
<td>1</td>
<td>1</td>
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<tr>
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<tr>
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<td></td>
</tr>
<tr>
<td>X2</td>
<td>2</td>
<td>1</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>X1( \frac{1}{2} )</td>
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<td></td>
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<td>1</td>
</tr>
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<td>3</td>
</tr>
<tr>
<td>TOTAL</td>
<td>3</td>
<td>3</td>
<td>5</td>
<td>5</td>
</tr>
</tbody>
</table>
SEEN ON THE TRUNK, SOMETIMES ON THE THORAX ALONE. A FEW MACULES MIGHT BE SEEN ON THE PROXIMAL PARTS OF THE LIMBS. IN ADDITION TO THE MACULAR ELEMENT A DEGREE OF ERYTHEMA OF THE TRUNK WAS NOTICED IN THREE CASES, IN TWO OF WHICH THE FACE AND NECK WERE ALSO FLUSHED. IN TWO OTHER CASES THE FACE AND NECK ALONE WERE FLUSHED. A WHITE LINE COULD USUALLY BE ELICITED IN THE AREAS OF FLUSHING.

SKETCHES TWO AND THREE SHOW MY IMPRESSION OF AN AVERAGE RASH.

THE SPLEEN.

SPLEENOMEGLY WAS PRESENT IN 50% OF MY CASES. THE SIGNIFICANCE OF THIS FINDING IS DIFFICULT TO ASSESS. MALARIA IS FREQUENTLY RESPONSIBLE, AND MALARIAL PARAZITAEMIA WAS PRESENT IN 33\% OF ALL CASES. IN SIXTEEN AUTOPSIES, THREE CASES WITH SPLENIC ENLARGEMENT HAD HAD MALARIAL PARAZITAEMIA DURING THEIR ILLNESS. THREE MORE WITH SPLENIC ENLARGEMENT PROBABLY HAD HAD MALARIA. FIVE CASES IN WHICH THERE WAS NO EVIDENCE OF MALARIA DURING LIFE OR AT AUTOPSY HAD SPLEENS OF NORMAL SIZE IN THREE CASES, AND IN ONE CASE ENLARGED BY ONE AND A HALF, IN ANOTHER CASE BY TWO. FIVE CASES DIAGNOSED AT AUTOPSY AND ON CLINICAL GROUNDS HAD SPLEENOMEGLY. THREE WERE ENLARGED ABOUT FIVE TIMES, ONE TWICE AND ONE ONE AND A HALF TIMES. THE LARGE SPLEENS REMAINED UNCHANGED
SKETCH NUMBER TWO.

Sketch showing a faint rash seen in a relatively fair oriental. The rash was not prominent, and unless looked for carefully, was easily missed.
SKETCH NUMBER THREE.

Sketch showing a faint typhus rash in a dark skinned oriental. The erythema was not so obvious. The papular element was more prominent than in the case seen in Sketch Number Two.
THROUGHOUT THE ILLNESS. THESE WERE PROBABLY CAUSED BY PREVIOUS MALARIA. THE IMPRESSION WAS GAINED THAT THERE WAS ONLY SLIGHT SPLENIC ENLARGEMENT IN THE CASES OF TYPHUS WHICH I SAW.

SEE TABLE TWENTY-ONE.

PROGRESS.

BY THE END OF THE FIRST WEEK OF FEVER THE PATIENT BECOMES QUITE DROWSY. IT IS VERY DIFFICULT TO GET A REPLY TO QUESTIONS AND IT IS THEN OFTEN ONLY "YES" OR "NO" TO LEADING QUESTIONS. DURING THE SECOND WEEK IN MILD CASES IMPROVEMENT MAY SET IN. IN THE MODERATE CASES THERE IS NO VERY MARKED CHANGE. SOME CASES HOWEVER BECOME SEVERELY ILL. THE PATHOLOGICAL PROCESS APPEARS TO CONCENTRATE EITHER IN THE LUNGS, WHICH BECOME MORE CONGESTED CAUSING CYANOSIS AND AIR HUNGER, OR IN THE BRAIN, PRODUCING IRRITABILITY AND RESTLESSNESS, ASSOCIATED WITH EVEN MORE INTENSE STUPOR. SLIGHT NECK RIGIDITY WAS SEEN IN THREE OF THESE CASES, AND IN ONE CASE MARKED NECK RIGIDITY ASSOCIATED WITH A POSITIVE KERNIG'S SIGN. IN THESE FEW CASES, THERE DID NOT SEEM TO BE ANY ASSOCIATION BETWEEN THE NECK RIGIDITY AND THE CEREBRO-SPINAL FLUID PRESSURE.

SEE TABLE TWENTY-TWO.
**TABLE TWENTY-TWO**

Table demonstrating that there is no obvious correlation between cerebro-spinal fluid pressure and the signs suggesting meningeal irritation (neck rigidity and positive Kernig's sign.)

<table>
<thead>
<tr>
<th>Signs Noted</th>
<th>C.S.F. Pressure in Millimeters of Water</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marked neck rigidity, Kernig's sign positive</td>
<td>200 M.M.</td>
</tr>
<tr>
<td>Slight neck rigidity, Kernig's sign negative</td>
<td>150 M.M.</td>
</tr>
<tr>
<td>Slight neck rigidity, Kernig's sign negative</td>
<td>175 M.M.</td>
</tr>
<tr>
<td>Slight neck rigidity, Kernig's sign negative</td>
<td>150 M.M.</td>
</tr>
<tr>
<td>No neck rigidity, Kernig's sign negative (not a fatal case)</td>
<td>338 M.M.</td>
</tr>
</tbody>
</table>

**TABLE TWENTY-THREE**

The percentage of cases with a rash in the various clinical groups.

<table>
<thead>
<tr>
<th>Very Mild</th>
<th>1 out of 11. 9.9%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate</td>
<td>5 out of 73. 88%</td>
</tr>
<tr>
<td>Severe</td>
<td>3 out of 9. 33.3%</td>
</tr>
<tr>
<td>Very Severe</td>
<td>1 out of 4. 25%</td>
</tr>
<tr>
<td>Fatal</td>
<td>5 out of 17. 29.4%</td>
</tr>
</tbody>
</table>
BY THE END OF THE SECOND WEEK AND MIDDLE OF THE THIRD WEEK MOST PATIENTS BEGIN TO RECOVER. GRADUALLY THE CHEST SIGNS BEGIN TO IMPROVE. THEY BECOME LESS DROWSY AND THE TEMPERATURE MAY BE LESS HIGH. THEY CAN BE MORE EASILY COAXED TO TAKE FLUIDS AND FOOD, AND IMPROVEMENT IS CONTINUOUS. THE SEVERE, AND VERY SEVERE CASES HOWEVER DO NOT IMPROVE. DURING THE SECOND WEEK THEIR CONDITION STEADILY DETERIORATES. AT THE END OF THE SECOND WEEK THEY MAY BE INCONTINENT OF URINE AND FAECES, VERY TOXIC AND RESTLESS, AND YET TURN THE CORNER IN A FEW DAYS. A RASH WAS MORE FREQUENTLY SEEN IN SEVERE, VERY SEVERE OR FATAL CASES.

SEE TABLE TWENTY-THREE.

THE FORM OF THE TEMPERATURE CHART.

IN GENERAL ONE MAY SAY THAT THE TYPHUS FEVER SEEN IN CALCUTTA WAS IN MOST CASES (86%), A CONTINUOUS FEVER, OFTEN WITH REMISSIONS, RARELY WITH INTERMISSIONS, AND FALLING BY LYSIS.

SEE TABLES TWENTY-FOUR, TWENTY-FIVE AND TWENTY-SIX.

IN FIVE CASES THE FALL OF THE TEMPERATURE WAS BY CRISIS. IN FIVE CASES THERE WAS AN APPARENT RELAPSE. IN FOUR CASES THE FEVER WAS IRREGULAR. IN THREE CASES THE FEVER WAS REMITTENT WITH A FALL BY LYSIS. IN THE FATAL CASES THE FEVER WAS CONTINUED IN SIX CASES,
## TABLE TWENTY FOUR
FORM OF TEMPERATURE CURVE IN FOUR CASES.

<table>
<thead>
<tr>
<th>Day of Disease</th>
<th>Temperature</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>104</td>
</tr>
<tr>
<td>7</td>
<td>103</td>
</tr>
<tr>
<td>9</td>
<td>102</td>
</tr>
<tr>
<td>11</td>
<td>101</td>
</tr>
<tr>
<td>13</td>
<td>100</td>
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<tr>
<td>15</td>
<td>99</td>
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<tr>
<td>17</td>
<td>98</td>
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<tr>
<td>19</td>
<td>98</td>
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<td>23</td>
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<tr>
<td>25</td>
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<table>
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<th>Temperature</th>
</tr>
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<td>2</td>
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<td>10</td>
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<td>20</td>
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<table>
<thead>
<tr>
<th>Day of Disease</th>
<th>Temperature</th>
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<tbody>
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</tr>
<tr>
<td>3</td>
<td>103</td>
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<tr>
<td>5</td>
<td>102</td>
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<td>7</td>
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<td>11</td>
<td>99</td>
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<td>13</td>
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<td>15</td>
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<td>17</td>
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<td>98</td>
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<tr>
<td>23</td>
<td>98</td>
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<table>
<thead>
<tr>
<th>Day of Disease</th>
<th>Temperature</th>
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</thead>
<tbody>
<tr>
<td>5</td>
<td>105</td>
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<td>7</td>
<td>104</td>
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<td>9</td>
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<td>21</td>
<td>98</td>
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<tr>
<td>23</td>
<td>98</td>
</tr>
</tbody>
</table>
### Table Twenty Five

**Further Examples of Form of Temperature Curves**

**Rash Lower Chest and Abdomen**

- Moderate Case.
- Blood Culture Sterile.
- Blood Culture Sterile (two).
- Rash on Trunk

**Blood Culture Sterile**

<table>
<thead>
<tr>
<th>Days</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
<th>Day 6</th>
<th>Day 7</th>
</tr>
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<tbody>
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**Blood Culture Sterile**

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**Blood Culture Sterile**

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</tr>
</tbody>
</table>
TABLE TWENTY SIX.
TABLE CLASSIFYING THE VARIOUS TYPES OF TEMPERATURE CURVES AND THE NUMBER OF CASES OF EACH

<table>
<thead>
<tr>
<th>Description</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAINLY CONTINUED FEVER WITH OCCASIONAL REMISSIONS OR INTERMISSIONS AND A FALL BY LYSIS</td>
<td>75</td>
</tr>
<tr>
<td>MAINLY CONTINUED FEVER WITH APPARENT RELAPSE</td>
<td>5</td>
</tr>
<tr>
<td>IRREGULAR FEVER</td>
<td>4</td>
</tr>
<tr>
<td>REMITTENT FEVER WITH FALL BY LYSIS</td>
<td>3</td>
</tr>
<tr>
<td>CASES SEEN DURING CONVALESCENCE</td>
<td>2</td>
</tr>
<tr>
<td>INTERMITTENT FEVER WITH FALL BY CRISIS</td>
<td>1</td>
</tr>
<tr>
<td>INTERMITTENT FEVER WITH FALL BY LYSIS</td>
<td>1</td>
</tr>
<tr>
<td>REMITTENT FEVER WITH INTERMISSIONS AND FALL BY LYSIS</td>
<td>1</td>
</tr>
</tbody>
</table>

FATAL CASES

<table>
<thead>
<tr>
<th>Description</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONTINUED FEVER WITH REMISSIONS</td>
<td>7</td>
</tr>
<tr>
<td>CONTINUED FEVER</td>
<td>6</td>
</tr>
<tr>
<td>CONTINUED FEVER, FALLING TOWARDS THE END</td>
<td>4</td>
</tr>
</tbody>
</table>
CONTINUED WITH REMISSIONS IN SEVEN CASES, AND
CONTINUED BUT FALLING TOWARDS THE END IN FOUR
CASES.

THE DURATION OF THE FEVER.

THE DURATION OF THE FEVER IN EIGHTY SIX PERCENT OF ALL CASES WAS BETWEEN NINE AND TWENTY-THREE DAYS. FORTY THREE PERCENT LASTED BETWEEN TWELVE AND SEVENTEEN DAYS. EXTREME LIMITS WERE FOUR DAYS AND THIRTY-THREE DAYS. THESE FIGURES CONFLICT WITH THOSE GIVEN IN MY ARTICLE (165) IN THE INDIAN MEDICAL GAZETTE. A FURTHER STUDY OF THE CASE HISTORIES AND THE TEMPERATURE CHARTS HAS LED ME TO MAKE SOME AMENDMENTS WHICH I THINK GIVE A MORE NEARLY CORRECT SET OF FIGURES.

SEE TABLE TWENTY-SEVEN.

A SUMMARY OF THE CLINICAL FINDINGS CONTRASTED WITH THOSE OF OTHER OBSERVERS IS SHOWN IN

TABLE TWENTY-EIGHT.

LABORATORY FINDINGS.

THE TOTAL WHITE CELL COUNT.

TOTAL WHITE CELL COUNTS WERERecorded
OF EIGHTY-FOUR PATIENTS. WHITBY AND BRITTON
(333) CONSIDER FIGURES OF 4,000 TO 11,000 TO
BE NORMAL LEVELS. FIFTY-EIGHT OR 69% OF THESE
EIGHTY-FOUR CASES WERE WITHIN THESE NORMAL
LIMITS. IN FORTY-SEVEN CASES ONLY A SINGLE
ESTIMATION WAS CARRIED OUT. IN ELEVEN OF THE
| FROM  | DURATION OF FEVER IN DAYS | 4 | 6-8 | 9-11 | 12-14 | 15 | 17 | 18-20 | 21 | 23-24 | 26 | 27 | 29 | 30 | 32 | 33 | 35 | ? |
|-------|---------------------------|---|-----|------|-------|----|----|-------|----|-------|----|----|----|----|----|----|----|----|---|
| JESSORE | NUMBER OF OXK CASES. | 1 | 2 | 6 | 11 | 7 | 2 | 1 | 1 | 1 |
|       | NUMBER OF OX19 CASES. | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
|       | NUMBER OF OXK CASES WITH CO-AGGLUTININS. | 2 | 6 | 3 | 4 | 2 | 2 | 2 | 2 | 2 |
|       | NUMBER OF FATAL OXK CASES | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
|       | NUMBER OF FATAL CASES DIAGNOSED ON CLINICAL AND AUTOPSY FINDINGS | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
| FROM  | NUMBER OF OXK CASES. | 1 | 2 | 4 | 6 | 4 | 5 | 3 | 1 | 1 |
| CALCUTTA | NUMBER OF OX19 CASES. | 2 | 3 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
|       | NUMBER OF OXK CASES WITH CO-AGGLUTININS. | 1 | 2 | 4 | 4 | 2 | 2 | 2 | 2 | 2 |
|       | NUMBER OF OX19 CASES WITH CO-AGGLUTININS. | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 |
|       | NUMBER OF FATAL OXK CASES. | 2 | 2 | 1 | 2 | 2 | 2 | 2 | 2 | 2 |
|       | NUMBER OF FATAL CASES DIAGNOSED ON CLINICAL AND AUTOPSY FINDINGS. | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
|       | DURATION OF ONE FATAL CASE WITH AGGLUTINATION OF OX2 AND CO-AGGLUTINATION OF OX19 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 2 | 2 |
TABLE TWENTY EIGHT

TABLE SHOWING THE MAIN CLINICAL FEATURES NOTED IN MY CASES, CONTRASTED WITH FINDINGS FROM OTHER REPORTS.

<table>
<thead>
<tr>
<th>SYMPTOMS</th>
<th>PACIFIC</th>
<th>BENGAL</th>
<th>ASSAM</th>
<th>CHIN HILLS</th>
<th>BURMA</th>
<th>BURMA</th>
<th>U.P.</th>
<th>INDIA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>GRIFFITHS (AMERICANS)</td>
<td>MEDEL (AMERICANS)</td>
<td>SANGER AND KAY (AUSTRALIANS)</td>
<td>LUK (INDIANS)</td>
<td>TATTERSALL (BRITISH)</td>
<td>TRENCH (BRITISH)</td>
<td>DESMUKH (EAST AFRICANS)</td>
<td>GURKESH SINGH (INDIANS)</td>
</tr>
<tr>
<td>Sudden Onset? Chill or Rigor</td>
<td>47%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>47%</td>
<td>47%</td>
<td>47%</td>
<td>47%</td>
</tr>
<tr>
<td>Eschar</td>
<td>80%</td>
<td>86%</td>
<td>34%</td>
<td>11%</td>
<td>8%</td>
<td>0</td>
<td>66%</td>
<td>0</td>
</tr>
<tr>
<td>Duration of Fever</td>
<td>9, 20 Days</td>
<td>20 Days</td>
<td>18 Days</td>
<td>18 Days</td>
<td>18 Days</td>
<td>18 Days</td>
<td>18 Days</td>
<td>18 Days</td>
</tr>
<tr>
<td>Headache</td>
<td>92%</td>
<td>92%</td>
<td>92%</td>
<td>92%</td>
<td>92%</td>
<td>92%</td>
<td>92%</td>
<td>92%</td>
</tr>
<tr>
<td>General Body Pain</td>
<td>13%</td>
<td>13%</td>
<td>13%</td>
<td>13%</td>
<td>13%</td>
<td>13%</td>
<td>13%</td>
<td>13%</td>
</tr>
<tr>
<td>Pain in Chest</td>
<td>8%</td>
<td>8%</td>
<td>8%</td>
<td>8%</td>
<td>8%</td>
<td>8%</td>
<td>8%</td>
<td>8%</td>
</tr>
<tr>
<td>Abdominal Pain</td>
<td>7%</td>
<td>7%</td>
<td>7%</td>
<td>7%</td>
<td>7%</td>
<td>7%</td>
<td>7%</td>
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</tr>
<tr>
<td>Suffused Eyes</td>
<td>38%</td>
<td>71%</td>
<td>17%</td>
<td>17%</td>
<td>17%</td>
<td>17%</td>
<td>17%</td>
<td>17%</td>
</tr>
<tr>
<td>Photophobia</td>
<td>45%</td>
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<td>45%</td>
<td>45%</td>
<td>45%</td>
<td>45%</td>
<td>45%</td>
<td>45%</td>
</tr>
<tr>
<td>Pharyngitis</td>
<td>8%</td>
<td>97%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>Cough</td>
<td>22%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>Signs of Bronchitis or Pneumonitis</td>
<td>51%</td>
<td>62%</td>
<td>68%</td>
<td>63%</td>
<td>63%</td>
<td>63%</td>
<td>63%</td>
<td>63%</td>
</tr>
<tr>
<td>Pulmonary Infarction</td>
<td>0</td>
<td>17%</td>
<td>17%</td>
<td>17%</td>
<td>17%</td>
<td>17%</td>
<td>17%</td>
<td>17%</td>
</tr>
<tr>
<td>Pneumonia or Lungs Complications</td>
<td>17%</td>
<td>0</td>
<td>10%</td>
<td>10%</td>
<td>10%</td>
<td>10%</td>
<td>10%</td>
<td>10%</td>
</tr>
<tr>
<td>Lymphadenitis</td>
<td>95%</td>
<td>95%</td>
<td>2 Cases</td>
<td>92%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>Rash Whites</td>
<td>35%</td>
<td>28%</td>
<td>78%</td>
<td>52%</td>
<td>64%</td>
<td>97%</td>
<td>97%</td>
<td>97%</td>
</tr>
<tr>
<td>Rash Natives</td>
<td>15%</td>
<td>31%</td>
<td>31%</td>
<td>31%</td>
<td>31%</td>
<td>31%</td>
<td>31%</td>
<td>31%</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>60%</td>
<td>60%</td>
<td>60%</td>
<td>60%</td>
<td>60%</td>
<td>60%</td>
<td>60%</td>
<td>60%</td>
</tr>
<tr>
<td>Temporary Deafness</td>
<td>20%</td>
<td>35%</td>
<td>35%</td>
<td>35%</td>
<td>35%</td>
<td>35%</td>
<td>35%</td>
<td>35%</td>
</tr>
<tr>
<td>Mental Disturbance</td>
<td>100%</td>
<td>12%</td>
<td>12%</td>
<td>12%</td>
<td>12%</td>
<td>12%</td>
<td>12%</td>
<td>12%</td>
</tr>
<tr>
<td>Mental Change Unlikely to Improve</td>
<td>1%</td>
<td>1%</td>
<td>1%</td>
<td>1%</td>
<td>1%</td>
<td>1%</td>
<td>1%</td>
<td>1%</td>
</tr>
</tbody>
</table>
FIFTY-EIGHT NORMALS, A REPEAT ESTIMATION WAS ALSO NORMAL. FOUR CASES WITH A LEUCOCYTOSIS HAD AT A PREVIOUS, OR A SUBSEQUENT EXAMINATION, A NORMAL COUNT. IN TWO FATAL CASES A NORMAL COUNT WAS FOLLOWED BY A LEUCOPENIA. A DEGREE OF LEUCOCYTOSIS WAS FOUND IN TWELVE CASES. FOUR OF THESE WERE FATAL CASES. A LEUCOPENIA WAS FOUND IN SINGLE ESTIMATIONS IN SEVEN CASES, ONE A FATAL CASE. IN ONE OTHER CASE A LEUCOPENIA SUBSEQUENTLY SHOWED AN INCREASE TO WITHIN NORMAL LIMITS. IN CONSIDERING THE COUNTS IN THE FATAL CASES IN TWELVE OF THESE IN WHICH ONE COUNT WAS CARRIED OUT, SEVEN WERE WITHIN NORMAL LIMITS. IN ONE CASE THERE WAS A LEUCOPENIA, IN FOUR CASES THERE WAS A LEUCOCYTOSIS. WHERE MORE THAN ONE COUNT WAS CARRIED OUT, ONE CASE SHOWED A NORMAL COUNT WITH A SUBSEQUENT LEUCOPENIA AND ONE CASE TWO NORMAL COUNTS WITH A SUBSEQUENT LEUCOPENIA.

SEE TABLE TWENTY-NINE

FROM THESE FIGURES ONE CANNOT MAKE ANY PRONOUNCEMENT ON THE ALTERATION, IF ANY, IN THE TOTAL WHITE COUNT IN THE COURSE OF THE DISEASE. THOSE CASES IN WHICH THERE WAS A LEUCOCYTOSIS, MORE FREQUENTLY SEEMED TO BE SEVERELY ILL, OR FATAL CASES.

SEE TABLES THIRTY, THIRTY-ONE, THIRTY-TWO, THIRTY-THREE AND THIRTY-FOUR.
**TABLE TWENTY NINE.**

Table showing the number of cases in which a total white cell count was estimated, grouped according to normality or otherwise of the count and the severity of the case.

<table>
<thead>
<tr>
<th>Type of Count</th>
<th>Number of Cases</th>
<th>Severity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Single Count</td>
<td>Repeated Counts</td>
</tr>
<tr>
<td><strong>Within Normal Limits</strong></td>
<td><strong>47</strong></td>
<td><strong>11</strong></td>
</tr>
<tr>
<td>Leucocytosis with normal count later</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Normal count with later Leucocytosis</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Leucocytosis</td>
<td><strong>12</strong></td>
<td></td>
</tr>
<tr>
<td>Leucopenia</td>
<td><strong>7</strong></td>
<td></td>
</tr>
<tr>
<td>Normal count with later Leucopenia</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Leucopenia with later normal count</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

Very mild 2
Moderate 46
Severe 6
Very severe 1
Fatal 9

Table showing the type of total white cell count seen in the fatal cases.

<table>
<thead>
<tr>
<th>Type of Count</th>
<th>Number of Cases</th>
<th>Severity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Single Count</td>
<td>Repeated Counts</td>
</tr>
<tr>
<td>Normal limits</td>
<td><strong>7</strong></td>
<td>2</td>
</tr>
<tr>
<td>Normal count with later Leucopenia</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Leucopenia</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Leucocytosis</td>
<td><strong>4</strong></td>
<td></td>
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</tbody>
</table>

**TABLE SHOWING THE NUMBER OF CASES IN WHICH A TOTAL WHITE CELL COUNT WAS ESTIMATED, GROUPED ACCORDING TO NORMALITY OR OTHERWISE OF THE COUNT AND THE SEVERITY OF THE CASE.**

<table>
<thead>
<tr>
<th>Type of Count</th>
<th>Number of Cases</th>
<th>Severity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Single Count</td>
<td>Repeated Counts</td>
</tr>
<tr>
<td><strong>Within Normal Limits</strong></td>
<td><strong>47</strong></td>
<td><strong>11</strong></td>
</tr>
<tr>
<td>Leucocytosis with normal count later</td>
<td>3</td>
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</tr>
<tr>
<td>Normal count with later Leucocytosis</td>
<td>1</td>
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</tr>
<tr>
<td>Leucocytosis</td>
<td><strong>12</strong></td>
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</tr>
<tr>
<td>Leucopenia</td>
<td><strong>7</strong></td>
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</tr>
<tr>
<td>Normal count with later Leucopenia</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Leucopenia with later normal count</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

Very mild 2
Moderate 46
Severe 6
Very severe 1
Fatal 9

<p>| DAY OF DISEASE | 7     | 8     | 9     | 10    | 11    | 12    | 13    | 14    | 15    | 16    | 17    | 18    | 19    | 20    | 21    | 22    | 23    | 24    | 25    | 26    | 27    | 30    |
|----------------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|
|                | 8500  | 9500  | 11000 | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 9500  | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 11000 | SEVERE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 14500 | (P.60% L.40%) |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 11000 | (R.74% L.34% E.2%) | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 6700  | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 9000  | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 14700 | (R.60% L.38% E.2%) | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 5000  | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 6700  | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 6000  | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 8400  | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 6200  | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 5900  | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 5200  | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 4500  | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 3500  | (P.60% L.34% M.6%) | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 8400  | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 9000  | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 4200  | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 5800  | SEVERE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 10000 | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 12800 | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | 3700  | MODERATE |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |
|                | MODERATE | 6000  |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |       |</p>
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<thead>
<tr>
<th>DAY OF THE DISEASE</th>
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<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
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<th>12</th>
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<th>17</th>
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<tbody>
<tr>
<td>11700</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>10400 (P.64% L.31% M.5%) SEVERE.</td>
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</tr>
<tr>
<td>8100 (P.70% L.24% M.6%)</td>
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</tbody>
</table>

**TABLE** SHOWING IN SEVENTEEN HIGH TITRE OXK CASES, THE DAY OF THE DISEASE ON WHICH TOTAL AND DIFFERENTIAL COUNTS WERE CARRIED OUT, THE ACTUAL COUNTS, AND THE SEVERITY OF THE CASE.
TABLE THIRTY TWO.

<table>
<thead>
<tr>
<th>DAY OF DISEASE</th>
<th>TOTAL AND DIFFERENTIAL COUNTS WERE CARRIED OUT, THE ACTUAL COUNTS AND THE SEVERITY OF THE CASE.</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>6000 MODERATE</td>
</tr>
<tr>
<td>6</td>
<td>5000</td>
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<tr>
<td>7</td>
<td>4000 MODERATE</td>
</tr>
<tr>
<td>8</td>
<td>3000 MODERATE</td>
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<td>9</td>
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<td>12</td>
<td>7000 SEVERE</td>
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<tr>
<td>13</td>
<td>7500 MODERATE</td>
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<td>14</td>
<td>7400 SEVERE</td>
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<td>16</td>
<td>7200 MODERATE</td>
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<td>7100 MODERATE</td>
</tr>
<tr>
<td>18</td>
<td>7000 MODERATE</td>
</tr>
<tr>
<td>19</td>
<td>6900 MODERATE</td>
</tr>
<tr>
<td>20</td>
<td>6800 MODERATE</td>
</tr>
<tr>
<td>21</td>
<td>6700 MODERATE</td>
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<td>22</td>
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<td>28</td>
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<tr>
<td>29</td>
<td>5900 MODERATE</td>
</tr>
<tr>
<td>30</td>
<td>5800 MODERATE</td>
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<table>
<thead>
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<th>DAY OF DISEASE</th>
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<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
<th>15</th>
<th>16</th>
<th>17</th>
<th>18</th>
<th>19</th>
<th>21</th>
<th>22</th>
<th>23</th>
<th>25</th>
</tr>
</thead>
<tbody>
<tr>
<td>6000</td>
<td>3600</td>
<td>(P.64% L.28% M.8%)</td>
<td>DEATH</td>
<td>15800</td>
<td>DEATH</td>
<td>4700</td>
<td>(P.80% L.16% M.4%)</td>
<td>DEATH</td>
<td>19200</td>
<td>(P.80% L.16% M.4%)</td>
<td>DEATH</td>
<td>15000</td>
<td>DEATH</td>
<td>6400</td>
<td>DEATH</td>
<td>7800</td>
<td>DEATH</td>
<td>9000</td>
</tr>
<tr>
<td>6800</td>
<td>(P.70% L.26% M.4%)</td>
<td>CLINICAL</td>
<td>6100</td>
<td>DEATH</td>
<td>9400</td>
<td>(P.71% L.26% M.3%)</td>
<td>DEATH</td>
<td>9100</td>
<td>7800</td>
<td>DEATH</td>
<td>16000</td>
<td>DEATH</td>
<td>5000</td>
<td>5200</td>
<td>DEATH</td>
<td>3900</td>
<td>DEATH</td>
<td>4600</td>
</tr>
</tbody>
</table>
THE TOTAL WHITE COUNT WAS OF SOME VALUE IN DIAGNOSIS, IN THAT IF IT WAS NORMAL IT DID NOT SUGGEST A PYOGENIC INFECTION ON THE ONE HAND, OR TYPHOID FEVER ON THE OTHER.

THE DIFFERENTIAL COUNT.

IT WAS NOT POSSIBLE TO CARRY OUT MORE THAN A FEW DIFFERENTIAL COUNTS; IN ALL ONLY FIFTEEN CASES WERE EXAMINED. WHITBY AND BRITTON (333) QUOTE AS NORMAL LIMITS THE FIGURES OF OSGOOD (230). WHITBY AND BRITTON (333) ALSO STATE THAT A LYMHPHOCYTOSIS IS REPORTED IN TYPHUS, PRESUMABLY IN EXANTHEMATIC TYPHUS.

IN EIGHT MODERATELY ILL CASES, A DEGREE OF NEUTROPHYL LEUCOCYTOSIS WAS SEEN IN TWO CASES AND IN ONE OF THESE CASES THERE WAS IN ADDITION AN INCREASE IN LYMPHOCYTES. NO SECONDARY INFECTION WAS ELICITED CLINICALLY TO EXPLAIN THE INCREASE.

IN THE FOUR FATAL CASES, A NEUTROPHYL LEUCOCYTOSIS WAS SEEN IN ONE CASE. AT AUTOPSY FIVE DAYS LATER NO OBVIOUS PYOGENIC INFECTION WAS FOUND TO ACCOUNT FOR THIS LEUCOCYTOSIS. TWO FATAL CASES HAD NORMAL DIFFERENTIAL COUNTS. IN ONE FATAL THERE WAS A LEUCOPENIA WITH DEPRESSION OF ALL TYPES OF WHITE CELL.

IN ONE SEVERE CASE THERE WAS A RELATIVE NEUTROPHYL LEUCOCYTOSIS, AND IN ANOTHER SEVERE CASE THE TOTAL AND DIFFERENTIAL COUNTS WERE
WITHIN NORMAL LIMITS.

THREE COUNTS IN ONE VERY SEVERE CASE SHOWED AN INCREASE IN NEUTROPHYS AND LYMPHOCYTES, AND IN ONE COUNT A RELATIVE INCREASE IN NEUTROPHYS, LYMPHOCYTES AND MONOCYTES. THIS CASE WAS COMPLICATED BY HAEMORRHAGE FROM THE BOWEL. THE TOTAL COUNT RETURNED TO NORMAL WITH RECOVERY.

ONE MAY SAY FROM THESE FIGURES THEREFORE, THAT IN THESE FEW CASES THERE DID NOT SEEM TO BE ANY OBVIOUS ALTERATION IN THE RELATIONSHIP OF THE VARIOUS WHITE CELLULAR ELEMENTS. INSUFFICIENT ESTIMATIONS WERE MADE TO DRAW ANY CONCLUSIONS IN SEVERELY ILL OR FATAL CASES. NOR WERE ANY EXAMINATIONS CARRIED OUT DURING THE FIRST FEW DAYS OF THE DISEASE. IN A PERSONAL COMMUNICATION PARKER (236) NOTED THAT IN SOME OF THE IMPHAL CASES OF SCRUB TYPHUS, SLIDES TAKEN EARLY IN THE DISEASE SHOWED NUMEROUS IMMATURE WHITE CELLS.

SEE TABLE THIRTY-FIVE.

EXAMINATION OF STOOLS AND URINE.

STOOLS AND URINE WERE CULTURED IN TWENTY-SIX INSTANCES. IN NO CASE WAS A BACILLUS PROTEUS ISOLATED.

BLOOD CULTURE.

BLOOD CULTURE WAS CARRIED OUT IN SIXTY-EIGHT CASES. IN FIFTY-TWO CASES CULTURE WAS STERILE. SIXTEEN SPECIMENS WERE CONTAMINATED.
TABLE THIRTY FIVE.

<table>
<thead>
<tr>
<th>DAY OF DISEASE</th>
<th>TOTAL COUNT</th>
<th>POLYMORPHS</th>
<th>LYMPHOCYTES</th>
<th>MONOCYTES</th>
<th>EOSINOPHILS</th>
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<td></td>
<td></td>
<td>MODERATE CASES</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>9</td>
<td>7400</td>
<td>4884 (66%)</td>
<td>2120 (30%)</td>
<td>148 (2%)</td>
<td>148 (2%)</td>
</tr>
<tr>
<td>12</td>
<td>3500</td>
<td>2100 (60%)</td>
<td>1190 (34%)</td>
<td>210 (6%)</td>
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</tr>
<tr>
<td>9</td>
<td>3500</td>
<td>2170 (62%)</td>
<td>1295 (37%)</td>
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<td>35 (1%)</td>
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<tr>
<td>16</td>
<td>8000</td>
<td>5840 (75%)</td>
<td>1440 (18%)</td>
<td>720 (9%)</td>
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<td>13</td>
<td>8100</td>
<td>5670 (70%)</td>
<td>1944 (24%)</td>
<td>486 (6%)</td>
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<tr>
<td>11</td>
<td>8400</td>
<td>6048 (72%)</td>
<td>1932 (23%)</td>
<td>336 (4%)</td>
<td>84 (1%)</td>
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<tr>
<td>10</td>
<td>11000</td>
<td>8140 (74%)</td>
<td>2640 (24%)</td>
<td>220 (2%)</td>
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<td>10</td>
<td>14700</td>
<td>8820 (60%)</td>
<td>5586 (38%)</td>
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<td>3080 (55%)</td>
<td>2240 (40%)</td>
<td>224 (4%)</td>
<td>56 (1%)</td>
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<td>10400</td>
<td>6656 (64%)</td>
<td>3224 (31%)</td>
<td>520 (5%)</td>
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<td>8480 (80%)</td>
<td>1378 (13%)</td>
<td>742 (7%)</td>
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<td>A VERY SEVERE CASE</td>
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<tr>
<td>9</td>
<td>14500</td>
<td>8700 (60%)</td>
<td>5800 (40%)</td>
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</tr>
<tr>
<td>14</td>
<td>14500</td>
<td>8700 (60%)</td>
<td>5075 (35%)</td>
<td>435 (3%)</td>
<td>145 (1%)</td>
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<tr>
<td>15</td>
<td>26800</td>
<td>1284 (48%)</td>
<td>9380 (35%)</td>
<td>4288 (16%)</td>
<td>268 (1%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FATAL CASES</td>
<td></td>
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</tr>
<tr>
<td>12</td>
<td>9400</td>
<td>6674 (71%)</td>
<td>2444 (26%)</td>
<td>282 (3%)</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>19200</td>
<td>15360 (80%)</td>
<td>3072 (16%)</td>
<td>768 (4%)</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>3600</td>
<td>2304 (64%)</td>
<td>1008 (28%)</td>
<td>288 (8%)</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>6800</td>
<td>4760 (70%)</td>
<td>1768 (26%)</td>
<td>272 (4%)</td>
<td></td>
</tr>
</tbody>
</table>
THE CEREBRO-SPINAL FLUID.

Greenfield and Carmichael (106) describe the changes in the cerebro-spinal fluid reported by observers of typhus in Europe. These are said to be, raised pressure except in moribund cases, cells moderately increased, protein slightly or moderately increased, the chlorides slightly reduced in some cases and increased in others, glucose normal or increased, urea constantly increased, and the Weil-Felix "frequently" or always positive, early in the disease.

1. C.S.F. PRESSURE.

Estimation of the cerebro-spinal fluid pressure was made with a spinal manometer in twenty-two cases. In fourteen cases the pressure was 175 mm. or over. The highest readings seen were in moderately ill cases. See Table Thirty-Six.

2. CELL CONTENT OF C.S.F.

A slight pleocytosis (5 to 9 per C.MM.) was seen in three out of six cases where the estimations were made. All were moderately ill. Two fatal cases had normal cell counts. See Table Thirty-Seven.

3. C.S.F. PROTEIN.

The protein was estimated in eleven cases. A slight increase to 35 mg.% was seen in two cases, one moderately ill and one fatal. A
**TABLE THIRTY SIX.**

Table showing the cerebro-spinal fluid pressures, the day of the disease on which these were estimated and the severity of the case.

<table>
<thead>
<tr>
<th>DAY OF DISEASE</th>
<th>6</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
<th>15</th>
<th>16</th>
<th>17</th>
<th>18</th>
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</thead>
<tbody>
<tr>
<td>180</td>
<td>VERY SEVERE</td>
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<tr>
<td>120</td>
<td>DEATH</td>
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<td>120</td>
<td>DEATH</td>
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<td>200</td>
<td>MODERATE</td>
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<td>320</td>
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<td>175</td>
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<tr>
<td>160</td>
<td>MODERATE</td>
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<tr>
<td>330</td>
<td>SEVERE</td>
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<td>388</td>
<td>MODERATE</td>
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<td>348</td>
<td>MODERATE</td>
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<td>150</td>
<td>DEATH</td>
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<tr>
<td>175</td>
<td>SEVERE</td>
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<td>MODERATE</td>
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<tr>
<td>360</td>
<td>MODERATE</td>
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<tr>
<td>150</td>
<td>DEATH</td>
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</tr>
</tbody>
</table>

**TABLE THIRTY SEVEN.**

Table showing the cerebro-spinal fluid cell count in six cases, the day of the disease on which the count was made and the severity of the case.

<table>
<thead>
<tr>
<th>DAY OF DISEASE</th>
<th>6</th>
<th>10</th>
<th>11</th>
<th>13</th>
<th>18</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>DEATH</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>MODERATE</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>MODERATE</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>MODERATE</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>MODERATE</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>DEATH</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
A more marked increase to 90 mg.% was seen in one severely ill case with considerable toxaemia and stupor, and clinically only a moderate degree of lung involvement. There was slight conjunctival congestion. In one fatal case there was a high protein reading due to meningeal congestion, the fluid being uniformly tinged with blood at puncture and being also blood stained at autopsy, with markedly congested meninges.

See Table Thirty-Eight.

4. C.S.F. Chloride.

The chloride content was estimated in eight cases. Slight reduction of chloride was seen in two moderately ill and two severe cases and one fatal case. It was within normal limits in two moderate cases and one fatal case.

See Table Thirty-Nine.

5. C.S.F. Urea.

The urea was estimated on four occasions. No conclusion can be drawn from the slightly high normal figures.

See Table Forty.

6. The Weil-Felix test in the C.S.F.

Seven estimations were carried out in six serologically proved cases, and in one clinical case. All were negative in all dilutions to Ox2, Ox19 and Oxk.
TABLE THIRTY EIGHT.
TABLE SHOWING THE ESTIMATIONS OF CEREBRO-SPINAL FLUID PROTEIN, AND THE DAY OF THE DISEASE ON WHICH ESTIMATIONS WERE MADE.

<table>
<thead>
<tr>
<th>DAY OF DISEASE</th>
<th>6</th>
<th>9</th>
<th>11</th>
<th>12</th>
<th>13</th>
</tr>
</thead>
<tbody>
<tr>
<td>25 MG. %</td>
<td>MODERATE</td>
<td>25 MG. %</td>
<td>MODERATE</td>
<td>20 MG. %</td>
<td>MODERATE</td>
</tr>
<tr>
<td>35 MG. %</td>
<td>MODERATE</td>
<td>30 MG. %</td>
<td>MODERATE</td>
<td>30 MG. %</td>
<td>SEvere</td>
</tr>
<tr>
<td>20 MG. %</td>
<td>SEvere</td>
<td>30 MG. %</td>
<td>SEvere</td>
<td>90 MG. %</td>
<td>SEvere</td>
</tr>
<tr>
<td>25 MG. %</td>
<td>MODERATE</td>
<td>35 MG. %</td>
<td>MODERATE</td>
<td>35 MG. %</td>
<td>FATAL</td>
</tr>
<tr>
<td>BLOOD STAINED C.S.F.</td>
<td>150 MG. %</td>
<td>FATAL</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

TABLE THIRTY NINE.
TABLE SHOWING THE ESTIMATIONS OF CEREBRO-SPINAL FLUID CHLORIDE AND THE DAY OF THE DISEASE ON WHICH ESTIMATIONS WERE MADE.

<table>
<thead>
<tr>
<th>DAY OF DISEASE</th>
<th>9</th>
<th>11</th>
<th>13</th>
<th>14</th>
<th>18</th>
</tr>
</thead>
<tbody>
<tr>
<td>700 MG. %</td>
<td>MODERATE</td>
<td>700 MG. %</td>
<td>MODERATE</td>
<td>720 MG. %</td>
<td>MODERATE</td>
</tr>
<tr>
<td>740 MG. %</td>
<td>MODERATE</td>
<td>700 MG. %</td>
<td>SEvere</td>
<td>720 MG. %</td>
<td>DEATH</td>
</tr>
<tr>
<td>680 MG. %</td>
<td>SEvere</td>
<td>700 MG. %</td>
<td>SEvere</td>
<td>700 MG. %</td>
<td>DEATH</td>
</tr>
</tbody>
</table>

TABLE FORTY.

<table>
<thead>
<tr>
<th>DAY OF DISEASE</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
</tr>
</thead>
<tbody>
<tr>
<td>26 MG. %</td>
<td>SEvere</td>
<td>26 MG. %</td>
<td>MODERATE</td>
<td>42 MG. %</td>
<td>CLINICAL</td>
<td>37 MG. %</td>
</tr>
<tr>
<td>26 MG. %</td>
<td>AGG. FOR OXID</td>
<td>MODERATE</td>
<td>DEATH</td>
<td>42 MG. %</td>
<td>CLINICAL</td>
<td>37 MG. %</td>
</tr>
</tbody>
</table>
**Table Forty One.**

Table showing the results of Weil-Felix tests of the cerebro-spinal fluid, the day of the disease on which the tests were carried out, the severity of the case and the blood Weil-Felix results. All the Weil-Felix results in the C.S.F. were negative.

<table>
<thead>
<tr>
<th>Degree of Severity of the Case</th>
<th>Day of Disease</th>
<th>Weil-Felix Results</th>
<th>Blood</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate</td>
<td>10</td>
<td>Negative</td>
<td>10th Day: 0X2 1/25 0X2 0 0XK 0 0XK 1/1000</td>
</tr>
<tr>
<td>Moderate</td>
<td>11</td>
<td>Negative</td>
<td>8th Day: 17th Day: 24th Day: 0X10 1/50 0X10 1/25 0X10 1/50 0X1k 1/25 0XK 1/50 0XK 1/250</td>
</tr>
<tr>
<td>Moderate</td>
<td>11</td>
<td>Negative</td>
<td>9th Day: 14th Day: 0X10 0 0X10 1/25 0X10 0 0XK 1/250</td>
</tr>
<tr>
<td>Moderate</td>
<td>11</td>
<td>Negative</td>
<td>11th Day: 18th Day: 20th Day: 0Xk 1/25 0Xk 1/50 0Xk 1/25</td>
</tr>
<tr>
<td>Moderate</td>
<td>13</td>
<td>Negative</td>
<td>10th Day: 14th Day: 0Xk 1/500 0Xk 1/1000</td>
</tr>
<tr>
<td>Severe</td>
<td>9</td>
<td>Negative</td>
<td>9th Day: 25th Day: 0X10 1/25 0X10 1/50 0Xk 1/50 0Xk 1/50</td>
</tr>
<tr>
<td>FATAL CASE Diagnosed on Clinical Grounds</td>
<td>6</td>
<td>Negative</td>
<td>Death on the Tenth Day.</td>
</tr>
</tbody>
</table>

**Table Forty Two.**

Table showing blood pressure readings, the day on which these were taken and the severity of the case.

<table>
<thead>
<tr>
<th>Day of Disease</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>16</th>
<th>17</th>
<th>18</th>
<th>19</th>
<th>23</th>
<th>27</th>
</tr>
</thead>
<tbody>
<tr>
<td>97%35</td>
<td></td>
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<td></td>
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<tr>
<td>99%65</td>
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<tr>
<td>91%70</td>
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<tr>
<td>99%70 Very Mild</td>
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<tr>
<td>100%70 Moderate</td>
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<td>110%70</td>
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<td>95%70</td>
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<tr>
<td>80%60</td>
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<td>95%70</td>
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<tr>
<td>10%60</td>
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<td>105%60</td>
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</tbody>
</table>
SEE TABLE FORTY-ONE.

SUMMARY OF CEREBRO-SPINAL FLUID FINDINGS.

The number of chemical and serological examinations is small. The findings suggest however that no such gross deviations from normal were present in my cases in Calcutta as are found in European typhus. There was very slight if any pleocytosis. Chloride was frequently reduced, but only below 700 mg.% in one case. Protein was frequently within normal limits. The urea was only estimated in four cases. Later observations on the kidney histology suggest that further investigations of renal function would be of value. The consistently negative Weil-Felix findings might be considered to be due to the observations being made too early in the course of the disease. The C.S.F. estimations were made round about the eleventh day. In one case however the blood titre for OXK was 1/500 three days before the C.S.F. examination was carried out. The cerebro-spinal fluid in my cases as far as one can judge therefore was not a help in diagnosis; except that a raised pressure was consistent with a diagnosis of typhus; and the absence of cells and organisms excluded meningitis.

The blood pressure.

Napier (213) considers that the systolic
PRESSURE IN INDIANS IS SEVEN TO TEN MILLIMETERS OF MERCURY LOWER THAN IN EUROPEANS OF A CORRESPONDING AGE GROUP, AND THE DIASTOLIC PRESSURE FIVE TO SEVEN MILLIMETERS LOWER. ESTIMATIONS ARE AVAILABLE FOR TEN OF MY CASES ONLY. THE FIGURES WERE LOW, AND IN VIEW OF THE GENERAL PROSTRATION AND WEAKNESS THIS WAS NOT SURPRISING.

SEE TABLE FORTY-TWO.

ESTIMATION OF HAEOMOGLOBIN.

ANAEMIA WAS NOT OFTEN SUSPECTED. HAEOMOGLOBIN WAS ONLY ESTIMATED ON FOUR OCCA-
SIONS. IN THREE CASES THE FIGURE, AS ESTIMATED IN GRAMS % BY SAHIL'S METHOD, WAS WITHIN THE NORMAL RANGE OF HAEOMOGLOBIN LEVEL FOR A COOLIE POPULATION IN BENGAL AND ASSAM AS SPECIFIED BY NAPIER AND DAS GUPTA (215). IN ONE CASE THE FIGURE WAS JUST BELOW THE NORMAL RANGE OF ONE SERIES OF INVESTIGATIONS BY NAPIER AND DAS GUPTA (215) IN ASSAM.

SERUM CALCIUM.

VAN MEERENDONK (318) HAS REPORTED LOW SERUM CALCIUM FIGURES IN CASES OF TYPHUS IN EUROPE. HIS FIGURES ARE OF THE ORDER OF 6.0 MG.%.

EVANS (78) CONSIDERS THAT DETERMINATIONS OF THE CALCIUM CONTENT OF SERUM ARE USELESS WITHOUT A SIMULTANEOUS ASSAY OF THE SERUM PROTEINS. UNFORTUNATELY SERUM PROTEIN ESTIMATIONS COULD NOT BE CARRIED OUT. THE FIGURES OBTAINED IN
FOURTEEN CASES EXAMINED SUGGESTED THAT THERE MAY BE A SLIGHT FALL IN SERUM CALCIUM, WITH A RISE DURING CONVALESCENCE, BUT THERE WAS NO CORRELATION BETWEEN THE SEVERITY OF THE DISEASE AND THE CALCIUM CONTENT OF THE BLOOD. ONE CASE WITH A RASH HAD A FIGURE OF 8.76 MG.%, NO LOWER THAN SEVERAL OTHER CASES IN WHICH NO RASH WAS SEEN.

SEE TABLE FORTY-THREE.

ESTIMATIONS IN OTHER DISEASES WERE OF THE SAME ORDER.

SEE TABLE FORTY-FOUR.

THE FINDINGS REQUIRE CORRELATION WITH THE SERUM PROTEIN LEVELS. THE FIGURES DO NOT SUGGEST THAT FIGURES OF THE ORDER OF 6.0 MG.% WERE PRESENT IN MY SERIES OF CASES.

COLD AGGLUTININS.

DURING AN INVESTIGATION OF PNEUMONIA IN INDIAN TROOPS, ESTIMATIONS OF COLD AGGLUTININS BY THE METHOD OF TURNER, NISNEWITZ, JACKSON AND BERNEY (315), WERE MADE ON SIX SEROLOGICALLY PROVED CASES OF TYPHUS AND ONE FATAL CASE IN WHICH SEROLOGICAL EVIDENCE WAS LACKING. WHITBY AND BRITTON (333) STATE THAT COLD AGGLUTININS ARE INCREASED IN PRIMARY ATYPICAL PNEUMONIA, TRYPANOSOMIASIS, SPIRILLOSIS, INFECTIOUS MONONUCLEOSIS, TROPICAL EOSINOPHILIA, SEVERE ANAEMIAS, MYELOMA AND CIRRHOSIS OF THE LIVER.
TABLE SHOWING SERUM CALCIUM LEVELS IN FOURTEEN CASES OF TYPHUS
WITH A NOTE OF THE DAY OF THE DISEASE ON WHICH ESTIMATIONS WERE MADE AND THE SEVERITY OF THE CASE.

<table>
<thead>
<tr>
<th>DAY OF DISEASE</th>
<th>8</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>20</th>
<th>21</th>
<th>24</th>
<th>27</th>
<th>28</th>
<th>54</th>
</tr>
</thead>
<tbody>
<tr>
<td>MILLISECONDS PER CENT</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>MODERATE</td>
<td>8.8</td>
<td>8.8</td>
<td>MODERATE 7.9</td>
<td>MODERATE 8.76</td>
<td>MODERATE 9.0</td>
<td>MODERATE 8.8</td>
<td>MODERATE 11.0</td>
<td>MODERATE 10.4</td>
<td>MODERATE 10.2</td>
<td>MODERATE 11.2</td>
</tr>
<tr>
<td>SEVERE</td>
<td>10</td>
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<td>VERY MILD</td>
<td>9</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RASH</td>
<td></td>
<td></td>
<td>MODERATE 8.2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

TABLE SHOWING SERUM CALCIUM LEVELS IN TEN CONTROL CASES OF BRONCHITIS AND PYREXIA OF UNKNOWN ORIGIN.

<table>
<thead>
<tr>
<th>DAY OF DISEASE</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>9</th>
<th>12</th>
<th>16</th>
<th>18</th>
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<tbody>
<tr>
<td>MILLISECONDS PER CENT</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BRONCHITIS (WHILE AFEBRILE)</td>
<td>11.0</td>
<td>10.4</td>
<td>8.4</td>
<td>9.0</td>
<td>9.2</td>
<td>8.9</td>
<td>8.2</td>
</tr>
<tr>
<td>TWO CASES OF BRONCHITIS (WHILE AFEBRILE)</td>
<td>10.4</td>
<td>9.0</td>
<td>8.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BRONCHITIS (WHILE AFEBRILE)</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>TWO CASES OF BRONCHITIS (WHILE AFEBRILE)</td>
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<td></td>
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<tr>
<td>TWO CASES OF PYREXIA OF UNKNOWN ORIGIN</td>
<td></td>
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<td>TWO CASES OF PYREXIA OF UNKNOWN ORIGIN</td>
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<tr>
<td>PYREXIA OF UNKNOWN ORIGIN</td>
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<td>PYREXIA OF UNKNOWN ORIGIN</td>
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<td></td>
</tr>
</tbody>
</table>
OF THE SEVEN CASES EXAMINED, IN THREE, NO COLD AGGLUTININS WERE DEMONSTRATED. IN ONE CASE AGGLUTINATION WAS PRESENT TO A TITRE OF 1/1. IN ONE CASE AGGLUTININS WERE PRESENT TO A TITRE OF 1/8 AND IN ANOTHER CASE TO A TITRE OF 1/16. IN A CASE CLINICALLY DIAGNOSED A TITRE OF 1/8 WAS RECORDED.

SEE TABLE FORTY-FIVE.

THE NUMBER OF OBSERVATIONS IS SMALL. THEY DO NOT SUGGEST HOWEVER THAT THERE WAS ANY MARKED INCREASE OF COLD AGGLUTININS IN MY CASES OF TROPICAL TYPHUS. AS IT IS A NON-SPECIFIC REACTION ACCORDING TO WHITBY AND BRITTON (333) ITS USE IN THE DIFFERENTIAL DIAGNOSIS OF PRIMARY ATYPICAL PNEUMONIA AND TYPHUS IS LIMITED.

COMPLICATIONS.

PAROTITIS.

IN TWO CASES A PAINFUL SWELLING OF THE PAROTID WAS OBSERVED. IT RESOLVED SLOWLY. SULPHAPYRIDINE DID NOT SEEM TO HAVE ANY BENEFICIAL EFFECT.

BLEEDING FROM THE GUMS.

THIS WAS A TROUBLESOME COMPLICATION IN THE FATAL CASE WHICH AGGLUTINATED OX2. THE BLEEDING STOPPED AFTER A TRANSFUSION OF WHOLE BLOOD.

HAEMORRHAGE FROM THE BOWEL.

SEVERE BLEEDING FROM THE BOWEL WAS SEEN
**TABLE FORTY FIVE.**

**TABLE SHOWING THE TITRE OF COLD AGGLUTININS IN SIX SEROLOGICALLY PROVED CASES OF TYPHUS AND ONE CLINICAL CASE.**

<table>
<thead>
<tr>
<th>Titre of Cold Agglutinins →</th>
<th>No Dilution</th>
<th>Dilution 1/4</th>
<th>Dilution 1/8</th>
<th>Dilution 1/16</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serologically Proved Cases</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<tr>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Clinical Case</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

**TABLE FORTY SIX.**

**TABLE SHOWING TEMPERATURE CHARTS, SHORT NOTES AND WEIL-FELIX RESULTS IN THREE VERY MILD CASES.**

<table>
<thead>
<tr>
<th>Day</th>
<th>Temperature</th>
<th>Short Notes</th>
<th>Blood Culture</th>
<th>Weil-Felix Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>02</td>
<td>105</td>
<td>Fever with rigors, backache, headache, looked ill, very weak. Few scattered rhonchi heard over both lungs. Convalescence rapid.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>07</td>
<td>105</td>
<td>Fever with rigor, lungs clear, marked general weakness. Rapid Convalescence.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>08</td>
<td>105</td>
<td>Fever with rigor, pain in the abdomen, general weakness, but general condition good. Lungs clear. Convalescence rapid.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
IN ONE CASE. LARGE DARK CLOTS WERE PASSED. A SMALL TRANSFUSION OF 200 CC. OF WHOLE BLOOD WAS FOLLOWED BY CESSATION OF THE BLEEDING IN SIX HOURS. THERE WAS A SMALL HAEMORRHAGE TWELVE HOURS LATER AND AGAIN THREE DAYS LATER. THE PATIENT, THOUGH VERY SEVERELY ILL, RECOVERED.

HAEMATURIA.

THIS COMPLICATION WAS SEEN IN TWO CASES DIAGNOSED AFTER AUTOPSY ON CLINICAL GROUNDS. BOTH HAD NUMEROUS SMALL PETECHIAL HAEMORRHAGES IN THE BLADDER WALL AND MUCOUS MEMBRANE.

SEE SKETCH NUMBERS TWENTY-EIGHT AND TWENTY-SEVEN.

HAEMOPTYSIS.

THREE PATIENTS, IN WHOM NO EVIDENCE OF TUBERCULOSIS COULD BE FOUND, COUGHED UP BLOOD-STAINED SPUTUM. IN ONE OF THESE CASES WHOSE CHEST WAS SCREENED A WEEK BEFORE HIS DEATH, THE RADIOLOGIST REPORTED THAT SHADOWS SUGGESTIVE OF TUBERCULOUS INFILTRATION WERE PRESENT. AT AUTOPSY NO EVIDENCE OF TUBERCULOSIS WAS FOUND, THOUGH THERE WAS THE TYPICAL PATCHY CONGESTION OF THE LUNGS SEEN IN TYPHUS.

MENTAL CONFUSION.

A COMMON OCCURRENCE WAS THAT STUPEROUS PATIENTS REFUSED FLUIDS OR REFUSED TO ALLOW THEIR TEMPERATURES TO BE TAKEN. TWO CASES HOWEVER SHOWED MARKED MENTAL CONFUSION WELL INTO CONVALESCENCE. BEFORE DISCHARGE HOWEVER, THEY HAD MENTALLY MADE A COMPLETE RECOVERY.
SEVERE EARACHE.

THIS WAS A COMPLAINT OF ONE PATIENT FOR WHICH THERE WAS NO OBVIOUS EXPLANATION; IT CLEARED UP IN A FEW DAYS.

DEAFNESS.

ONE PATIENT BECAME DEAF AND REMAINED SO WELL INTO CONVALESCENCE. HE MADE A COMPLETE RECOVERY.

VARIATIONS FROM THE AVERAGE CASE.

MILD CASES.

ELEVEN CASES WERE CLASSIFIED AS VERY MILD FORMS OF THE DISEASE. SOME CASES WERE ABLE TO WALK SLOWLY AROUND THE WARD AND EAT A FAIRLY LIBERAL DIET EVEN WHILE FEBRILE.

SEE TABLE FORTY-SIX.

SEVERE AND VERY SEVERE CASES.

THE SEVERE AND VERY SEVERE CASES DID NOT MARKEDLY DIFFER FROM THE MODERATE CASES, EXCEPT IN THE GREATER DEGREE OF TOXAEMIA. A RASH WAS MORE COMMONLY SEEN. THE DURATION OF THE FEVER WAS LONGER. A DRAMATIC RECOVERY WAS FREQUENTLY SEEN IN MANY CASES WHO LOOKED DESPERATELY ILL.

SEE TABLE FORTY-SEVEN.

THE FATAL CASES.

THE CAUSE OF DEATH SEEMED TO BE TOXAEMIA AND ANOXAEMIA. BAD PROGNOSTIC SIGNS WERE INCREASING CONGESTION OF THE LUNGS WITH INCREASING ANOXAEMIA, AND INCREASING STUPOR WITH ANOXAEMIA. INCONTINENCE OF EITHER URINE OR FAECES WAS ALSO OF ILL OMEN AS ALSO WAS EXTREME
### TABLE FORTY SEVEN

**Table showing temperature charts with short notes and Weil-Felix results in two severe, and two very severe cases.**

#### Fever, Marked Lung Signs, Eyes Congested, Tox and Ill. Slow Recovery.

**Severe Case.**

#### Rash.

**Blood Culture Sterile.**

**Fever and Chill, Chest Full of Moist Sounds, Considerable Prostration, Temporary Mental Upset, and Slow Recovery.**

**Severe Case.**

#### Blood Films Negative for Malaria.

**Fever with Headache, Backache and Cough; Congestion of Lungs Present, Eyes Injected, Drowsy, Eyes Injected, Muttering Delirium, Later a Rash Present.**

**Haemorrhage from Bowel Controlled by 200 cc. of Blood IV. Slow Recovery. Very Severe Case.**

#### Blood Films Negative for Malaria.

**Fever, Cough and Headache, Chest Signs Second Week. Incontinence of Urine, Slow Recovery.**

**Very Severe Case.**

---

**C.S.F.,** 

**Stool and Urine Culture Negative.**

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| TABLE FORTY EIGHT |
| SHOWING TEMPERATURE CHARTS, WEIL FELIX RESULTS AND SHORT NOTES IN FOUR FATAL CASES |

1. **RASH.**
   - Blood culture sterile
   - Admitted as measles, delirious eyes injected, soreness of lips, flushing of skin of trunk with macular rash, lungs congested, muttering delirium, I.V. saline given, incontinence of urine, deterioration of circulation, death on 15th day.

2. **Fever with rigor, with increasing congestion of lungs, deepening coma, cyanosis, restlessness, death on 18th day.**
   - Blood culture sterile
   - Onset, fever with rigor, on admission, drowsy and ill. Coarse crepitations and rhonchi all over chest, no impairment of percussion note. Muttering delirium, twitching of fingers, slight stiffness of neck, circulatory failure, death 16th day. Strain isolated on 12th day.

3. **Sudden onset of fever.**
   - Blood culture sterile
   - Faint rash
   - Marked congestion of lungs, deepening coma, death on 24th day.
RESTLESSNESS.

SEE TABLE FORTY-EIGHT.

ISOLATION OF A RICKETTSIAL STRAIN FROM A FATAL CASE.

MAJOR M.T. PARKER, R.A.M.C. (239) (240),
while in charge of the district laboratory in Calcutta isolated a strain from one of my cases, who contracted the disease in Calcutta. Major Parker was fortunate in having the advice of Dr. S.R. Savor on technical details. Five cc. of blood was allowed to clot. The ground up clot was injected into a guinea pig and passaged into white mice, in the peritoneal exudate of which rickettsiae were seen. The strain was maintained in mice and forwarded to Cairo. It is of interest that in the case from which this strain was isolated, no necrotic lesion was seen, no rash observed and the Weil-Felix on the sixteenth day of the disease (the day of his death) was only 1/125 OXK. The autopsy findings of patchy haemorrhagic areas in the lungs, haemorrhagic areas in the caecum and small subarachnoid haemorrhages in the pia-arachnoid with general congestion of the brain, were parallel with the findings in the other fatal cases.

TREATMENT.

Symptomatic treatment was given. In this connection McRobert's (184) article published in 1925 seemed to me to contain the essential points.
IN GENERAL TREATMENT TO BE OBSERVED BY THOSE IN CHARGE OF TYPHUS PATIENTS. THE TREATMENT OF RESTLESSNESS, IF NECESSARY USING MORPHINE TO PRODUCE SLEEP; THE TREATMENT OF DEHYDRATION BY ADEQUATE MEASURES; AND THE RELIEF OF INCREASED CEREBRO-SPINAL FLUID PRESSURE BY LUMBAR PUNCTURE ARE PROCEDURES WHICH RESULT IN OBVIOUS CLINICAL IMPROVEMENT. MCROBERT RECOMMENDED INTRAVENOUS SODIUM CITRATE FOR THREATENED THROMBOSIS IN HIS CASES. THROMBOSIS WAS NOT A COMPLICATION IN MY SERIES. DEHYDRATION AND ITS TREATMENT IN THE TROPICS HAS BEEN STUDIED BY MARRIOTT (174). ANDREW (4) HAS STRESSED THE DANGER OF INTRAVENOUS FLUID IN CASES WHERE THE FLUID BALANCE IS DISTURBED. I HAVE FOUND HOWEVER THAT THE FEVERED INDIAN PATIENTS ARE MOST RELUCTANT TO DRINK. THIS IS IN MANY CASES DUE TO EXCESSIVE CHLORIDE LOSS. THE REPLACEMENT OF FLUID AND CHLORIDE BY AN INTRAGASTRIC DRIP THROUGH A RYLES TUBE IS A SATISFACTORY METHOD BY WHICH FLUID MAY BE ADMINISTERED. IN 1943 HOWEVER, RYLES TUBES WERE NOT AVAILABLE IN CALCUTTA, BUT I FOUND THAT INTRAVENOUS NORMAL GLUCOSE SALINE IN QUANTITIES OF THREE, FOUR, OR EVEN SIX OR EIGHT PINTS COULD BE GIVEN DAILY WITH BENEFIT TO STUPOROUS PATIENTS DURING THE HOT WEATHER. I AM IN AGREEMENT WITH MEGAW (195) THAT THE CLINICAL CONDITION MUST BE TAKEN INTO ACCOUNT
IN ESTABLISHING THE SUITABLE FLUID INTAKE.

NO SPECIFIC TREATMENT HAS SO FAR BEEN UNIVERSALLY ACCEPTED. PARA AMINO BENZOIC ACID HAS BEEN USED FOLLOWING ANIMAL EXPERIMENTS. MURRAY, ZARAFONETIS AND SNYDER (209) USING GEBRELLES, SHOW THAT THERE IS CONSIDERABLE PROTECTION GIVEN BY PARA AMINO BENZOIC ACID TO INTRA-PERITONEAL INJECTIONS OF LETHAL DOSES OF RICKETTSIA ORIENTALIS. SNYDER AND ZARAFONETIS (292) AND HAMILTON (110) HOWEVER DO NOT FIND ANY MARKED EFFECT IN THE PREVENTION OF GROWTH OF R. ORIENTALIS IN INFECTED YOLK SACS, THOUGH SNYDER AND ZARAFONETIS CONSIDERED THERE WAS A RETARDATION OF THE GROWTH OF R. RICKETTSIA AND LESS SO R. PROWAZEKI AND R. PROWAZEKI VAR. MOOSERI. ANIGSTEIN AND BADGER (7) FOUND A 100% RECOVERY RATE IN GUINEA PIGS IN WHICH PARA AMINO BENZOIC ACID WAS GIVEN PROPHYLACTICALLY FROM THE DAY OF INNOCULATION. THESE EXPERIMENTAL FINDINGS SUGGEST THAT THE DRUG HAS A PROPHYLACTIC VALUE. CLINICALLY HOWEVER, TIERNEY (311) REPORTS STRIKINGLY GOOD RESULTS OBTAINED FROM THE TREATMENT OF EIGHTEEN SCRUB TYPHUS CASES IN ASSAM, AS COMPARED WITH SIXTEEN CONTROLS. SMALLER DOSES USED BY SMITH (290) IN THE TREATMENT OF MURINE TYPHUS DID NOT SHOW SUCH SPECTACULAR RESULTS, THOUGH THE DURATION OF THE FEVER WAS SHORTER. ROSE, DUANE AND FISCHELL (267) ALSO WITH SMALLER DOSES REPORT BENEFICIAL
EFFECTS IN ONE CASE OF ROCKY MOUNTAIN SPOTTED FEVER. FURTHER INVESTIGATIONS WILL NO DOUBT BE OF INTEREST. ZARAFONETIS, SNYDER AND MURRAY (353) CONSIDER THAT THE IMMUNITY FOLLOWING PARA AMINO BENZOIC ACID THERAPY IN EXPERIMENTAL TSUTSUGAMUSHI DISEASE SUGGESTS THAT THE ACTION OF THE DRUG "IS TO INHIBIT THE GROWTH", RATHER THAN TO KILL THE RICKETTSIAE. NO SUPPLIES OF THIS DRUG WERE AVAILABLE FOR THE TREATMENT OF MY CASES. NOR WAS PENICILLIN AVAILABLE. REPORTS ON EXPERIMENTAL WORK ON ANIMALS BY TOPPING (314) AND A CLINICAL REPORT BY KLEIN (138) SUGGEST THAT PENICILLIN IS OF NO VALUE IN TSUTSUGAMUSHI DISEASE (SCRUB TYPHUS). VAN MEERENDONK'S (318) FIGURES FOR SERUM CALCIUM HAVE BEEN MENTIONED. HE TREATED HIS CASES WITH ATEBRIN AND CALCIUM. NO FIGURES FOR CONTROLS WERE QUOTED. MEGAW (194) POINTS OUT THAT THERE IS NO EVIDENCE THAT ATEBRIN HAS ANY EFFECT ON THE CASUAL RICKETTSIA. MANY OF MY CASES WERE HAVING MEPACRINE FOR CONCOMITANT MALARIA WITH NO OBVIOUS IMPROVEMENT. WEIGL'S (327) METHOD OF INFECTING LICE BY RECTAL INNOCULATION WITH RICKETTSIAE PROWAZEKI, WAS THE FIRST STEP IN THE PRODUCTION OF A VACCINE. SPENCER AND PARKER (299) FIRST DEMONSTRATED THAT IT WAS POSSIBLE TO PRODUCE IMMUNITY TO ROCKY MOUNTAIN SPOTTED FEVER BY THE USE OF A KILLED VIRUS.

IMPROVED METHODS OF CULTIVATION AS THE
COMBINED AGAR-TISSUE-CULTURE AND A YOLK SAC CULTURE OF ZINNSER (357) AND HIS CO-WORKERS, AND THE INSUFFLATION OF MICE LUNGS WITH RICKETTSIAE BY THE METHOD OF DURRAND AND SPARROW (72) HAVE ENABLED VACCINE TO BE PRODUCED ON A SUFFICIENTLY LARGE SCALE TO PROTECT ARMIES IN THE FIELD DURING THE SECOND WORLD WAR. UNTIL THAT WAR CREATED THE DEMAND, NO VACCINE WAS AVAILABLE FOR TSUTSUGAMUSHI FEVER. THE BRITISH GOVERNMENT DECIDED TO PROCEED WITH THE MANUFACTURE OF A SCRUB TYPHUS VACCINE ON THE LINES DESCRIBED BY FULTON AND JOYNER (100). A COMPLETE LABORATORY WAS READY WITHIN 109 DAYS OF THE DECISION TO START THE OPERATION.

DETAILS OF THE PRODUCTION ARE GIVEN BY BUCKLAND AND CO-WORKERS (39). COTTON RATS WERE THE ANIMALS USED. THREE OUT OF SIXTY WORKERS BECAME INFECTED. ALL HAD BEEN VACCINATED.

THE WAR WITH JAPAN ENDED BEFORE THE VACCINE COULD BE GIVEN AN EXTENDED FIELD TRIAL. WALKER (325) DESCRIBES THE EFFECT OF THIS PARTICULAR VACCINE WHEN GIVEN TO SIXTEEN CASES WHO WERE THOUGHT TO BE INCUBATING THE DISEASE AND CONSIDERS THAT IN THOSE WHO HAD TWO OR THREE DOSES OVERLAPPING THE INCUBATION PERIOD THE SEVERITY OF THE DISEASE WAS CONSIDERABLY MODIFIED. HE MAKES NO COMMENT ON THE EFFICACY OF THE VACCINE IN 2500 TROOPS WHO WERE
INNOCULATED. WITH THE END OF HOSTILITIES TROOPS WERE NOT COMPELLED TO COME INTO CONTACT WITH THE JUNGLE IN QUITE THE SAME WAY AS DURING HOSTILITIES, AND THERE HAS NOT THEREFORE BEEN THE OPPORTUNITY TO TEST THE VACCINE UNDER EQUIVALENT CONDITIONS OF EXPOSURE.

THE ACTION OF METHYLENE BLUE IN EXPERIMENTAL SCRUB TYPHUS HAS BEEN STUDIED BY MCLIMANS AND GRANT (182) WITH ENCOURAGING RESULTS. STEELE AND COLLABORATORS (301) SUGGEST HOWEVER THAT METHYLENE BLUE ALONE HAS SO FAR PROVED INEFFECTUAL IN THE TREATMENT OF SCRUB TYPHUS.

THE DIFFICULTIES IN OBTAINING MATERIALS FOR FIELD TRIALS OF A SCRUB TYPHUS VACCINE ARE VERY CONSIDERABLE. THE NEW ANTIBIOTIC SUBSTANCES MAY VERY WELL PROVIDE A SPECIFIC WHICH WILL RENDER TREATMENT EFFECTIVE.

**PATHOLOGY OF PERSONAL CASES.**

OUT OF THE SEVENTEEN FATAL CASES, AUTOPSIES WERE CARRIED OUT ON SIXTEEN. ELEVEN OF THESE CASES WERE SEROLOGICALLY POSITIVE TO PROTEUS OXK WITH NO CROSS-AGGLUTINATION. THE OTHER FIVE CASES WERE CONSIDERED TO BE TYPHUS ON CLINICAL GROUNDS AND WITH CONSISTENT POST-MORTEM APPEARANCES. THE MORBID ANATOMICAL APPEARANCES OF THE ELEVEN SEROLOGICALLY POSITIVE CASES WILL BE DESCRIBED.
MORBID ANATOMY.

ESCHAR

IN NO CASE WAS ANY DERMAL LESION SEEN TO SUGGEST THE ESCHAR DESCRIBED AND ILLUSTRATED BY LEWTHWAITE AND SAVOOR (149) OR BLAKE, MAXCY, SADUSK, KOHLS AND BELL (24).

RASH.

IN TWO CASES A FAINT MACULAR RASH WAS SEEN ON THE TRUNK. IN ONE CASE, IN WHICH DURING LIFE AN ERYTHEMATOUS BLUSH WAS SEEN OVER THE TRUNK, A BLOTCHY RASH WITH A PETECHIAL ELEMENT WAS SEEN OVER THE BUTTOCKS AND THIGHS AT AUTOPSY.

RESPIRATORY TRACT.

THE TONSILS AND UPPER RESPIRATORY TRACT WERE NORMAL. THE TRACHEA AND LARGE BRONCHI WERE CONGESTED IN FIVE CASES.

SEE SKETCH NUMBER FOUR.

THE PLEURA.

OCCASIONAL PLEURAL ADHESIONS WERE SEEN. THEY WERE ASSOCIATED AS A RULE WITH CONGESTION OF THE ADJACENT LUNG SUBSTANCE, AND WERE SOMETIMES EASILY BROKEN DOWN BUT SOMETIMES WERE SEPARATED WITH DIFFICULTY.

SEE SKETCHES FIVE AND EIGHT.

RECENT FINE PLEURAL ADHESIONS WERE SEEN IN FIVE CASES, IN SOME CASES INTERLOBAR. THEY COULD BE SEPARATED EASILY.
SKETCH SHOWING CONGESTION OF THE TRACHEA AND BRONCHI. THERE IS ALSO PATCHY CONGESTION OF THE LUNG PARENCHYMA WITH INTERLOBAR ADHESIONS.
SKETCH NUMBER FIVE.

Sketch of lung showing thickening of the pleura with congestion of the adjacent lung parenchyma, with scattered patches of congestion in the lung and with subpleural haemorrhages. These appear to be recent.
SEE SKETCH SIX

OLD GENERALISED ADHESIVE PLEURSY WAS SEEN IN ONE CASE. SUBPLEURAL HAEMORRHAGES WERE SEEN IN FIVE CASES. THESE HAEMORRHAGES VARIED IN SIZE FROM 2 MM. TO 10 MM. IN DIAMETER, AND APPEARED TO BE FAIRLY RECENT LESIONS, THOUGH SOME WERE DARK IN COLOUR.

SEE SKETCHES FIVE, SIX AND SEVEN.

THESE MAY BE COMPARED WITH A SIMILAR APPEARANCE SEEN IN THE LUNGS OF A FATAL CASE OF PARATYPHOID C. FEVER. IN THAT CASE NUMEROUS SMALL PUNCTATE HAEMORRHAGES WERE SEEN OVER A LOCALISED AREA OF ONE LOBE.

SEE SKETCH NUMBER NINE.

THE LUNGS.

IN TWO CASES THE LUNGS WERE CREPITANT AND NO OBVIOUS CHANGE WAS NOTED. IN THE OTHER NINE CASES THERE WAS A VARYING DEGREE OF INCREASE IN FIRMNESS, DUE TO CONGESTION; IN NO CASE WAS PNEUMONIA OR BRONCHO-PNEUMONIA SUSPECTED FROM THE NAKED EYE APPEARANCE. IN EIGHT CASES THE CONGESTION WAS OF A PECULIAR PATCHY CHARACTER. THERE WAS A UNIFORM PLUM-COLOURED CONGESTION, APICAL IN ONE CASE, BASAL IN THE OTHERS, AND IN THESE CONGESTED AREAS DARKER PATCHES OF MORE INTENSE CONGESTION WERE SEEN VARYING IN SIZE FROM ABOUT 3 MM. TO 10 MM. THERE WAS NO OBSERVED RELATIONSHIP BETWEEN
SKETCH NUMBER SIX.

SKETCH OF A LUNG SHOWING FINE INTERLOBAR ADHESIONS AND SUBPLEURAL HAEMORRHAGES OF VARIOUS SIZES. THESE APPEAR TO BE RECENT.

SKETCH NUMBER SEVEN

SKETCH OF A LUNG SHOWING SUBPLEURAL HAEMORRHAGES. THESE ARE SOMewhat LARGER THAN THOSE DEMONSTRATED IN THE PREVIOUS SKETCHES.
SKETCH NUMBER EIGHT

Adherent pleura with marked congestion of the underlying lung. There is also recent apical pleurisy.

SKETCH NUMBER NINE

Punctate pleural haemorrhages seen in a case of paratyphoid C fever. Compared with haemorrhages seen in cases of typhus, these are essentially similar, though smaller and more circumscribed.
THESE MORE DEEPLY CONGESTED AREAS AND BLOOD VESSELS OR BRONCHI.

SEE SKETCHES FOUR, FIVE, TEN AND ELEVEN.

THESE MAY BE COMPARED WITH A SECTION OF THE LUNG IN PARATYPHOID C. FEVER IN WHICH THERE WERE SUBPLEURAL HAEMORRHAGES AS SEEN IN SKETCH NINE. THE PATCHY CONGESTION IS LESS OBVIOUS IN THE CASE OF PARATYPHOID C.

SEE SKETCH TWELVE.

URINARY TRACT.

KIDNEY.

IN ALL CASES THE CAPSULES STRIPPED READILY LEAVING A SMOOTH SURFACE. IN TWO CASES THE APPEARANCES SUGGESTED CLOUDY SWELLING. IN SEVEN CASES THE KIDNEYS APPEARED CONGESTED, MAINLY IN THE CORTEX. NO CHANGES WERE NOTED IN THE URETERS OR BLADDER IN THE SEROLOGICALLY PROVED CASES.

CARDIO-VASCULAR SYSTEM.

PERICARDIUM.

THERE WAS NO SIGN OF PERICARDIAL DISEASE EXCEPT IN ONE CASE WHERE THE PARITETAL AND VISCERAL PERICARDIUM WERE MATTED TOGETHER BY OLD ADHESIONS. IT WAS POSSIBLE HOWEVER TO SEPARATE THEM.

MYOCARDIUM.

IN TWO CASES THE MYOCARDIUM WAS
SKETCH NUMBER TEN.

PATCHY CONGESTION OF A LUNG AS WAS COMMONLY SEEN IN MY CASES.

SKETCH NUMBER ELEVEN.

A LARGE PATCH OF CONGESTION AS SEEN ON SECTION OF THE LUNG SHOWN IN SKETCH NUMBER EIGHT.
SKETCH NUMBER TWELVE.

A SKETCH OF A SECTION OF THE LUNG SHOWN IN SKETCH NUMBER NINE.
THE PATCHY CONGESTION IS NOT OF THE SAME DEGREE AS IS USUALLY SEEN IN THE PATCHY CONGESTION SEEN IN CASES OF TYPHUS.

SKETCH NUMBER THIRTEEN.

HAEMORRHAGES IN THE PIA-ARACHNOID OF THE FRONTAL AREA OF THE BRAIN.
CONSIDERED TO BE SOFT WITH IN ONE CASE DILATION OF THE RIGHT HEART, IN THE OTHER CASE, GENERAL PALLOR OF THE MUSCLE. IN THE OTHER CASES NO OBVIOUS ABNORMALITY WAS SEEN.

ENDOCARDIUM.
THERE WERE NO OBVIOUS ENDOCARDIAL LESIONS.

CENTRAL NERVOUS SYSTEM.

THE MENINGES.
CONGESTION OF THE MENINGEAL VESSELS WAS CONSIDERED TO BE PRESENT IN TWO CASES, AND IN TWO CASES A FEW RECENT SMALL HAEMORRHAGES WERE SEEN IN THE PIA-ARACHNOID.

SEE SKETCH THIRTEEN.
IN ONE CASE, NOT IN THIS SERIES, SEEN AT DIMAPUR IN ASSAM, THERE WAS EXTREME CONGESTION OF THE SURFACE OF THE BRAIN WITHOUT CONGESTION ON SECTION.

SEE SKETCHES FOURTEEN AND FIFTEEN.
MARKED OEDEMA OF THE PIA-ARACHNOID WAS SEEN ONCE.

SEE SKETCH SIXTEEN.

THE BRAIN.
OEDEMA OF THE BRAIN WAS NOTED IN THREE CASES. THE BRAIN WHEN SECTIONED SHOWED NO ABNORMALITY, OTHER THAN PALLOR IN THOSE CASES OEDEMA WAS PRESENT.

SEE SKETCH SEVENTEEN.
SKETCH NUMBER FOURTEEN

EXTREME CONGESTION OF THE BRAIN SEEN IN A CASE OF SCRUB TYPHUS. COMPARE WITH THE CONGESTION SEEN IN MALARIA.

SKETCH NUMBER FIFTEEN.

SECTION OF THE BRAIN SEEN IN SKETCH FOURTEEN. COMPARE WITH THE SECTION OF A BRAIN FROM A CASE OF CEREBRAL MALARIA.
SKETCH NUMBER SIXTEEN.

OEDEMA OF THE PIA-ARACHNOID.

SKETCH NUMBER SEVENTEEN.

SECTION OF BRAIN SEEN IN SKETCH SIXTEEN, THERE IS A CERTAIN DEGREE OF PALLOR.
THERE IS NO CONGESTION OF VESSELS SEEN.
IN THE TWO CASES WHERE THERE WAS CONGESTION OF THE MENINGEAL VESSELS THERE WAS NOT ANY MARKED INCREASE IN GREYNESS OF THE CORTEX ON SECTION AS IS SEEN IN CEREBRAL MALARIA, NOR WAS THERE THE MARKED CONGESTION OF THE VESSELS OF THE GREY AND WHITE MATTER SEEN IN THE SAME DISEASE. SKETCHES OF GROSS EXTERNAL APPEARANCE AND SECTION OF A BRAIN FROM A CASE OF CEREBRAL MALARIA ARE SHOWN FOR COMPARISON.

SEE SKETCHES EIGHTEEN AND NINETEEN.

ALIMENTARY TRACT.

STOMACH AND DUODENUM.


SEE SKETCHES TWENTY AND TWENTY-ONE.

SMALL BOWEL.

CONGESTION OF THE MUCOUS MEMBRANE AND HÄMORRHAGIC SPOTS WERE SEEN IN THREE CASES. THESE SPOTS WERE USUALLY ABOUT 3-4 MM. IN DIAMETER AND IN NEARLY ALL CASES WERE BRIGHT RED SUGGESTING THAT THEY WERE RECENT, THOUGH IN ONE
Sketch of a Brain from a case of Cerebral Malaria seen in Shillong, in Assam. Note the intense congestion and the marked cyanosis characteristic of the condition.

Sketch Number Nineteen

Sketch of a section of the brain seen in Sketch Number Eighteen. Note that there is a marked distinction between the white and grey matter. This was not seen in my cases of typhus. Note also the pin points of congested vessels in the white matter.
CONGESTION OF THE MUCOUS MEMBRANE OF THE STOMACH.

CONGESTION OF THE MUCOUS MEMBRANE OF THE STOMACH WITH ULCERATION ALONG THE RUGAE.
CASE THERE WERE ABOUT TWENTY-FOUR SPOTS MOSTLY BRIGHT RED, BUT BEING IN SOME INSTANCES A DARKER RED COLOUR. SOME OF THESE WERE MORE ON THE MUCOUS SURFACE, A FEW SEEMED INTRA-MURAL, AND ONE WAS ON THE PERITONEAL SURFACE OF THE GUT. IN TWO CASES THERE WAS CONGESTION OF THE ILEO-CAECAL VALVE.

SEE SKETCHES TWENTY-TWO, TWENTY-THREE AND TWENTY-FOUR.

IN ONE CASE THERE WAS GENERALISED CONGESTION OF THE PERITONEUM OF THE SMALL GUT, WITH NO OBVIOUS LESION OF THE MUCOUS MEMBRANE.

LARGE BOWEL.

THERE WAS A GENERAL CONGESTION OF THE MUCOUS MEMBRANE OF THE CAECUM IN SIX CASES. IN ONE CASE THIS WAS SLIGHT, IN THE OTHER FIVE IT WAS MARKED. TWO OF THESE WERE ASSOCIATED WITH ULCERATION WHICH WAS CONSISTENT WITH AMOEBAIC INFECTION, THOUGH DURING LIFE THERE HAD BEEN NO SYMPTOMS OF DYSENTRY. IN TWO CASES THERE WERE AREAS OF CONGESTION IN THE MUCOUS MEMBRANE OF THE ASCENDING, DESCENDING AND PELVIC COLON.

SEE SKETCHES TWENTY-FOUR AND TWENTY-FIVE.

IN ONE CASE THERE WAS CONGESTION OF THE PERITONEUM OF THE LARGE BOWEL ONLY, WITH NO OBVIOUS ABNORMALITY OF ITS MUCOUS MEMBRANE.

PANCREAS.

NO OBVIOUS LESIONS WERE SEEN.
SKETCH NUMBER TWENTY TWO.

SKETCH SHOWING CONGESTION OF THE TERMINAL ILEUM AND CONGESTION OF THE ILEO-CAECAL VALVE.

SKETCH NUMBER TWENTY THREE.

SKETCH SHOWING AN INTERMURAL HAEMORRHAGE IN THE SMALL BOWEL WITH CONGESTION OF THE MUCOUS MEMBRANE AND PERITONEUM.
SKETCH NUMBER TWENTY FOUR,

CONGESTION OF THE CAECUM AND OF THE ILEO-CAECAL VALVE. THREE ULCERS ARE SEEN, PRESUMABLY SILENT AMOEbic ULCERS.

SKETCH NUMBER TWENTY FIVE.

CONGESTION OF THE LARGE BOWEL.

SKETCH NUMBER TWENTY SIX.

HAEMORRHAGE IN CORTEX OF KIDNEY.
LIVER.

In three cases the liver was soft, pale and swollen and in three cases soft and pale, and in one case pale. In one case it was swollen and soft and of a uniform dark greenish red colour. In one case there was some mottling of the liver. In the other two cases there was no very obvious naked eye change.

GALL BLADDER.

In one case there was a slight congestion of the gall bladder.

SPLEEN.

The spleen was found to be enlarged in nine cases. In five cases it was increased up to double the average size, in one case three times, in one case four times and in two cases five times the average size. In all but one case the pulp was firm, and the splenic architecture recognisable. In one case the pulp was soft and diffluent. In this case splenic smears were negative for malarial parasites, but the patient had had treatment, including intravenous quinine for malignant tertian malaria.

LYMPH GLANDS.

The mesenteric lymph glands were enlarged in three cases, measuring three-quarters of an inch to one inch in diameter.
All three cases had congestion of the mucous membrane or intra mural haemorrhages. One of these cases had in addition enlargement of the crevical glands to the size of kidney beans, and slight enlargement of the axillary glands. On section the glands were soft and pink or purplish in colour. There was no obvious necrosis. In one case there was an enlarged gland in the hilum of a lung. The lung showed patchy congestion.

Appearances of cases not serologically proved.

When considering whether a case is likely to be typhus, Lewthwaite's (147) observation may be kept in mind, that there is "no single consistent feature on naked eye examination peculiar to tropical typhus. The gross findings consisted rather of an aggregation of a few pathological changes, usually only of a slight degree, each of which is to be observed individually and some of them collectively in the morbid anatomy of other diseases". Three of the five cases diagnosed without serological proof died on the tenth, eleventh and thirteenth days of their illness respectively when the Weil-Felix has sometimes not yet risen to diagnostic titre. In a case which died on the fourteenth day, oxk on the
TWELFTH DAY WAS 1/50. IN THE OTHER TWO CASES CONTAMINATED SPECIMENS PRECLUDED SEROLOGY. THEY ALL SHOWED LESIONS OF THE SAME NATURE AS DESCRIBED FOR THE SEROLOGICALLY PROVED CASES, AND IN ADDITION TWO CASES SHOWED EPITHELIAL AND MURAL HAEMORRHAGES IN THE BLADDER, AND ONE OF THESE SHOWED A DIFFUSE HAEMORRHAGE IN THE CORTEX OF ONE KIDNEY.

SEE SKETCHES TWENTY-SIX, TWENTY-SEVEN AND TWENTY-EIGHT.

IN ONE CASE THERE WAS SUCH MARKED MENINGEAL CONGESTION THAT THE CEREBRO SPINAL FLUID WAS BLOOD STAINED. THIS I ALSO SAW LATER IN A SEROLOGICALLY PROVED CASE AT DIMAPUR IN ASSAM OF WHICH I MADE A SKETCH AT THE TIME.

SEE SKETCH FOURTEEN.

MORBID HISTOLOGY.

HISTOLOGICAL EXAMINATION OF FORMALIN FIXED TISSUES STAINED WITH HAEMATOXYLIN AND EOSIN IN CALCUTTA REVEALED, GENERALLY SPEAKING, NO GROSS HISTOLOGICAL CHANGES WHICH MIGHT BE CONSIDERED TYPICAL OF TYPHUS. THOUGH THE LUNG APPEARANCES WERE THEN GENERALLY CONSIDERED TO BE DUE TO THE PRIMARY INFECTION, IT WAS FELT THAT THE TYPHUS LESION MIGHT BE PRESENT IN THE LUNG CAPILLARIES, PRODUCING THE PATCHY
SKETCH NUMBER TWENTY SEVEN

A CASE DIAGNOSED ON CLINICAL AND AUTOPSY FINDINGS.

HAEMORRHAGES IN THE WALL OF THE BLADDER.

SKETCH NUMBER TWENTY EIGHT.

SKETCH OF THE INTERIOR OF THE BLADDER OF THE CASE SHOWN IN SKETCH TWENTY SEVEN SHOWING HAEMORRHAGIC SPOTS AND ONE SMALL ULCER.
HAEMORRAGES, SEEN NAKED EYE. THE RETICULO-ENDOTHELIAL HYPERPLASIA OF THE ENLARGED LYMPH NODES WAS SEEN, THE GENERAL CONGESTION OF MANY ORGANS WAS NOTED, AND THE APPEARANCES CONSISTENT WITH A FEBRILE TOXAEARIA WAS NOT UNEXPECTED, BUT NOWHERE WAS ANY LESION OF A VESSEL SUCH AS HAVE BEEN DESCRIBED IN EXANTHEMATIC TYPHUS AND IN ROCKY MOUNTAIN SPOTTED FEVER.

SECTIONS WERE STAINED WITH GIEMSA AND A SEARCH MADE FOR RICKETTSIAE. WHEN NOTHING WAS SEEN WHICH COULD WITH CERTAINTY BE RECOGNISED AS SUCH, FURTHER ATTEMPTS TO IDENTIFY RICKETTSIAE IN THE TISSUES WERE ABANDONED AS TOO TIME-CONSUMING, THE ARMY AUTHORITIES WERE THEN MORE CONCERNED WITH AN ATTEMPT TO ISOLATE A STRAIN OF RICKETTSIA FROM WHICH A VACCINE MIGHT BE PRODUCED TO PROTECT THE TROOPS FROM CONTRACTING THE DISEASE IN THE FORWARD AREAS. THREE STRAINS WERE ISOLATED, TWO FROM IMPHAL CASES AND ONE FROM A CASE OF MINE IN CALCUTTA AS I HAVE ALREADY MENTIONED.

AT A LATER DATE I SELECTED A NUMBER OF SECTIONS FOR STUDY FROM THREE CASES WHICH HAD BEEN SEROLOGICALLY PROVED. I WAS FORTUNE ENOUGH TO BE ABLE TO HAVE SOME OF THESE SECTIONS RE-STAINED WITH GIEMSA BY MR. T. C. DODDS OF THE PATHOLOGICAL DEPARTMENT, EDINBURGH.
UNIVERSITY. THE LARGE AMOUNT OF PIGMENT IN MANY OF THE SLIDES WAS COMMENTED ON BY THOSE WHO EXAMINED THEM. IT WAS CONSIDERED THAT THIS PIGMENT MIGHT BE LARGELY DUE TO THE ACTION OF THE FORMALIN IN WHICH THE TISSUES WERE FIXED. NO RICKETTSIAE WERE SEEN IN RESTAINED SECTIONS. THIS IS NOT SURPRISING, AS ALLEN AND SPITZ (2) IN AN EXTENSIVE SEARCH OF CONSIDERABLE MATERIAL WERE ABLE TO DEMONSTRATE RICKETTSIAE IN ONLY ONE SECTION, A SECTION OF AN ESCHAR. THEY NOTE THAT SPECIAL METHODS OF FIXING, STAINING AND CUTTING OF SECTIONS IS REQUIRED AND ADD THAT "AT BEST, THE UNMASKING OF RICKETTSIAE IN THE TISSUES OF SCRUB TYPHUS IS CURRENTLY AN UNRELIABLE PROCEDURE".

THE LUNGS.

FIVE SECTIONS WERE EXAMINED OF WHICH FOUR WERE RESTAINED WITH GIEMSA. THE NAKED EYE APPEARANCE OF PATCHY CONGESTION IS ALSO SEEN MICROSCOPICALLY. IN SOME FIELDS AREAS OF APPARENTLY NORMAL LUNG TISSUE ARE SEEN, WITH ADJOINING AREAS OF CONGESTION OF THE ALVEOLAR WALL AND A CELLULAR EXUDATE IN THE ALVEOLI. THE CELLS OF THE ALVEOLAR WALLS ARE SWOLLEN.

THE ALVEOLAR EXUDATE CONSISTS OF LARGE CELLS WITH ROUND OR OVAL NUCLEI. SOME CELLS ARE SMALLER WITH A DEEPLY STAINING NUCLEUS. THE NUCLEI OF THE LARGER CELLS HAVE A RECTICULAR NUCLEAR STRUCTURE. A FEW OF THE VERY
LARGE CELLS ARE MULTINUCLEATED. THE CYTOPLASM OF THESE CELLS IS USUALLY FAINTLY NEUTROPHILIC. SOME HOWEVER TAKE UP MORE OF THE ACID DYE. IN SOME AREAS QUITE A NUMBER OF RED BLOOD CELLS ARE SEEN IN THE ALVEOLI. ONLY AN OCCASIONAL NEUTROPHYL POLYMORPH IS SEEN AND THERE IS NO FIBRIN.

SEE MICROPHOTOGRAPHS ONE AND TWO AND SKETCH TWENTY-NINE.

SOME ALVEOLI ARE SEEN CONTAINING A SCANTY EXUDATE. IN SOME PLACES THE ALVEOLAR WALLS ARE SWOLLEN WITH NO ALVEOLAR EXUDATE.

SEE SKETCHES THIRTY AND THIRTY-ONE.

WHERE THE EXUDATE IN THE ALVEOLI IS SCANTY THE CELLS ARE THE SAME AS THOSE SEEN IN MARKED CONGESTED AREAS BUT ARE PERHAPS THE LARGER CELLS; AND LARGE MULTINUCLEATED CELLS ARE MORE OFTEN SEEN.

SEE SKETCH THIRTY-TWO.

IN A CONSIDERABLE AREA OF ONE SECTION THE ALVEOLI ARE FILLED WITH AN UNIFORM TRANSLUCENT EXUDATE IN WHICH THERE ARE A FEW POLYMORPHS AND NUMEROUS DIPLOCOCCI ARE SEEN, SUGGESTIVE OF AN EARLY TERMINAL PNEUMONIC PROCESS.

SEE MICROPHOTOGRAPH THREE.

A FRANK PNEUMONIC PROCESS IS NOT APPARENT IN ANY OF THE SECTIONS.

ERYTHROPHAGOCYTOSIS AND CYTOPHAGOCYTOSIS IS
MICROPHOTOGRAPH ONE.

Area with considerable area with cellular exudate in the alveoli

MICROPHOTOGRAPH TWO.

Type of cellular exudate in alveoli

H&E. SECTION OF LUNG X70

H&E. SECTION OF LUNG X750

Thickened alveolar wall

Binuclear cell
SKETCH NUMBER TWENTY NINE.

STAIN H & E.  

CONGESTED ALVEOLAR WALL  

BINUCLEATED CELL  

EXUDATE OF CELLS WITH ROUND OVAL OR RENIFORM NUCLEI.

SKETCH NUMBER THIRTY.

STAIN H & E.  

ALVEOLAR CONGESTION WITH A SCANTY ALVEOLAR EXUDATE.
**Sketch Number Thirty One.**

*Stain: Giemsa*

Swollen alveolar wall with no exudate in the alveolus.

**Sketch Number Thirty Two.**

*Stain: H&E*

Multinucleated cell in albuminous exudate.
MICROPHOTOGRAPH THREE.

A section of a lung showing alveoli filled with an albuminous exudate, in which many polymorphonuclear cells are seen.

MICROPHOTOGRAPH FOUR

Section of kidney showing cloudy swelling, oedema of the interstitial tissues and an interstitial cellular reaction. The cells are large, with round or oval faintly staining nuclei.
SEEN ON VERY RARE OCCASIONS. THE CELLULAR REACTION IS NOT RELATED TO EITHER BRONCHI OR BLOOD VESSELS AS FAR AS COULD BE SEEN.

URINARY TRACT.

KIDNEY.

THREE SECTIONS WERE EXAMINED, ONE FROM EACH CASE. ONE OF THESE WAS RESTAINED WITH GIEMSA. ONE SECTION SHOWS VERY MARKED CLOUDY SWELLING, PRINCIPALLY OF THE CONVOLUTED TUBULES, LESS SO OF HENLE'S LOOPS. THERE IS A SLIGHT DEGREE OF OEDema OF THE INTERSTITIAL TISSUES AND OF THE GLOMERULAR TUFTS. IN SOME CASES THE LATTER ALMOST FILLS THE CAPSULAR SPACE BUT THERE DOES NOT SEEM TO BE NUCLEAR PROLIFERATION. THERE IS NO THICKENING OF BOWMAN'S CAPSULE.

SEE SKETCHES THIRTY-THREE, THIRTY-FOUR AND THIRTY-FIVE.

ANOTHER SECTION SHOWS CLOUDY SWELLING OF THE TUBULES AND HENLE'S LOOPS. THERE IS OEDema OF THE INTERSTITIAL TISSUE. THE GLOMERULI SHOW NUCLEAR PROLIFERATION BUT SEEM PATENT AS RED BLOOD CELLS ARE SEEN IN THE GLOMERULAR CAPILLARIES. NO EPITHELIAL CRESCENTS ARE SEEN. CERTAIN AREAS IN THE CORTEX ARE THE SEAT OF A PERIGLOMERULAR REACTION. A FEW GLOMERULI ARE ADHERENT TO BOWMAN'S CAPSULE. VARYING DEGREES OF OBLITERATION OF THE GLOMERULI ARE SEEN.
SKETCH NUMBER THIRTY THREE.

STAIN
H & E.

SKETCH SHOWING CLOUDY SWELLING OF THE TUBULES. THERE IS A DEGREE OF INTERSTITIAL OEDEMA AND OEDEMA OF THE GLOMERULI.

SKETCH NUMBER THIRTY FOUR.

H & E.

SKETCH SHOWING OEDEMA OF GLOMERULI WITH NO APPRECIABLE NUCLEAR PROLIFERATION.
SKETCH NUMBER THIRTY FIVE
H&E.  x 425

SKETCH FROM THE SAME SECTION AS NUMBERS THIRTY THREE AND THIRTY FOUR. THERE IS APPARENT OEDEMA OF THIS GLOMERULAR TUFT BUT NO OBVIOUS NUCLEAR PROLIFERATION.

SKETCH NUMBER THIRTY SIX
H&E.  x 425

SKETCH FROM THE SAME SECTION AS NUMBERS THIRTY THREE, THIRTY FOUR AND THIRTY FIVE. THERE IS CONGESTION OF THE GLOMERULAR TUFT.
SEE MICROPHOTOGRAPHS FOUR AND FIVE AND SKETCHES THIRTY-SIX AND THIRTY-SEVEN.

THE CELLS IN THE AREAS OF REACTION CONSIST OF DEGENERATED TUBULAR CELLS AND LARGE CELLS WITH OVAL RENIFORM OR IRREGULARLY SHAPED NUCLEI.

SEE SKETCH THIRTY-EIGHT.

IN THE SAME SECTION, AREAS OF REACTION ARE ALSO SEEN IN THE INTERSTITIAL TISSUE AT THE JUNCTION OF THE CORTEX AND MEDULLA. THESE CELLS ALSO HAVE OVAL RENIFORM OR IRREGULARLY SHAPED NUCLEI.

SEE MICROPHOTOGRAPH SIX

A THIRD SECTION RESTAINED WITH GIEMSA SHOWED ON THE WHOLE NORMAL GLOMERULI. IN THE INTERSTITIAL TISSUE BETWEEN THE TUBULES THERE ARE HERE AND THERE SMALL AREAS OF CELLULAR REACTION. THE CELLS ARE MEDIUM SIZED WITH ROUND OR OVAL NUCLEI WITH DELICATE RETICULUM, AND ALSO SMALLER CELLS WITH MORE DEEPLY STAINING NUCLEI AND LESS CYTOPLASM. SOMETIMES THESE CELLULAR REACTIONS, SEEMED RELATED TO A BLOOD VESSEL, BUT OFTEN NOT OBVIOUSLY SO. A FEW GLOMERULI ARE SEEN TO BE ADHERENT AT ONE POINT TO A THICKENED BOWMAN'S CAPSULE. SURROUNDING THE ATTACHMENT ARE A FEW EPITHELOID CELLS.

SEE MICROPHOTOGRAPH SEVEN.
Area of focal interstitial reaction around glomerulae. There is nuclear proliferation of the tuft which however still seems patent in many instances. There is considerable pericapsular fibrosis in some cases with adhesion of the tuft at one point.

Microphotograph six.

Intense cloudy swelling of tubules with interstitial cellular reaction at junction of cortex and medulla.
Sketch Number Thirty Seven.

Sketch from the same sections as numbers thirty three, thirty four, thirty five and thirty six, showing nuclear proliferation in a glomerular tuft but apparently patent capillaries. There is degeneration of the tubules.

Sketch Number Thirty Eight.

Sketch showing the type of focal cellular reaction round a glomerulus. The cells are large with oval or irregular nuclei.
Giemsa  
MICROPHOTOGRAPH SHOWING A GLOMERULAR TUFT ADHERANT TO BOWMAN'S CAPSULE AND A CELLULAR REACTION AROUND THE ADHESION.

H&E  
MICROPHOTOGRAPH OF HEART MUSCLE SHOWING AN INTERSTITIAL CELLULAR REACTION. IN THIS CASE THERE WERE DENSE ADHESIONS BETWEEN THE PARietal AND VISCERAL PERICARDIUM AND THE LESION WAS PROBABLY AN OLD RHEUMATIC FOCUS.
NO RICKETTSIAE COULD BE SEEN IN THIS SECTION. NO ABNORMALITIES ARE SEEN IN THE VESSELS OF ANY OF THE SECTIONS.

HEART.

THREE SECTIONS MAINLY CONSISTING OF HEART MUSCLE WERE AVAILABLE FROM TWO CASES. ONE OF THESE WAS RE-STAINED WITH GIEMSA. IN NONE OF THESE SECTIONS WERE ANY VERY OBVIOUS CHANGES SEEN. EXCEPT IN ONE SECTION, WHERE ONE FIELD SHOWS AT ONE POINT AN INTERSTITIAL CELLULAR REACTION. THE CELLS ARE MOSTLY OVAL WITH A DELICATE RETICULAR NUCLEUS, AND NEUTROPHILIC CYTOPLASM. A FEW CELLS WITH ROUND DENSE NUCLEI AND A SMALL AMOUNT OF CYTOPLASM ARE PROBABLY LYMPHOCYTES, WHILE A FEW ELONGATED CELLS WITH OVAL NUCLEI RESEMBLE FIBROBLASTS.

SEE MICROPHOTOGRAPH EIGHT.

IN THIS CASE THE VISCERAL AND PARIETAL PERICARDIUM WERE ADHERENT AND COULD ONLY BE SEPARATED WITH DIFFICULTY. I CONSIDER THAT THIS IS DUE TO PREVIOUS RHEUMATIC INFECTION. ONE SECTION RE-STAINED WITH GIEMSA SHOWED NOTHING ABNORMAL AND NO RICKETTSIAE WERE SEEN.

LYMPHOID TISSUE.

THREE SECTIONS WERE AVAILABLE, ONE FROM AN ENLARGED HILAR GLAND. THERE IS
CONGESTION OF THE VESSELS. THERE IS NO MARKED HYPERPLASIA OF THE LYMPH NODES, NOR IS THERE ANY OBVIOUS NECROSIS OF THE LYMPHOID TISSUE. THE LYMPH SINUSES ARE CONGESTED AND CONTAIN MANY LARGE CELLS. ERYTHROPHAGOCYTOSIS AND CYTOPHAGOCYTOSIS IS FREQUENTLY SEEN.

SEE MICROPHOTOGRAPHS NINE AND TEN.

SPLLEEN.

THREE SECTIONS WERE EXAMINED, ONE BEING RESTAINED WITH GIEMSA. THERE DOES NOT SEEM TO BE ANY MARKED INCREASE IN THE LYMPHOID TISSUE, BUT AMONGST THE LYMPHATIC CELLS, LARGER OVAL CELLS, WITH FAINT STAINING CYTOPLASM AND OVAL RETICULAR NUCLEI ARE SEEN IN SMALL NUMBERS. OCCasionally A LARGE CELL WITH A CIRCULAR DARK NUCLEUS AND ACIDOPHILIC CYTOPLASM IS SEEN. THERE IS ENGORGEMENTS OF THE SINUSOIDS. ERYTHROPHAGOCYTOSIS IS SEEN. MULTINUCLEATED LARGE CELLS ARE SEEN ALSO. BOTH SECTIONS SHOW LARGE NUMBERS OF ERYTHROCYTES IN THE SPLENIC PULP.

SEE MICROPHOTOGRAPH ELEVEN.

GASTRO INTESTINAL TRACT.

THREE SECTIONS FROM VARIOUS PARTS OF THE SMALL BOWEL OF ONE CASE SHOWED LOCAL CONGESTION IN THE MUCOUS LAYER, LESS SO IN THE SUBMUCOUS LAYERS.

SEE MICROPHOTOGRAPHS TWELVE AND THIRTEEN.
SECTION OF LYMPH GLAND SHOWS NO GROSS DEVIATION IN GLAND STRUCTURE.

SECTION OF LYMPH GLAND SHOWING CONGESTED LYMPH SINUSES. THE CELLS VARY IN SIZE AND SHAPE. ERYTHROPHAGOCYTOSIS IS SEEN.
SECTION OF SPLEEN SHOWING TYPES OF CELLS SEEN IN THE SPLENIC PULP

SECTION OF SMALL BOWEL SHOWING AN AREA OF LOCAL CONGESTION OF THE MUCOUS AND SUBMUCOUS LAYERS. ALSO IN THE SUBMUCOUS LAYER A SOLITARY FOLLICLE IS SEEN. LARGER CELLS WITH OVAL RETICULAR NUCLEI MAY BE SEEN WITH A HIGHER POWER, AMONG THE LYMPHOCYTES.
THE CELLS WERE OF MEDIUM SIZE WITH HOMOGENEOUS NEUTROPHYL CYTOPLASM AND ROUND, OVAL, OCCASIONALLY RENIFORM NUCLEI. THE NUCLEI WERE OF A LOOSE RETICULAR STRUCTURE WITH PERIPHERAL CHROMATIN. IN ONE SECTION A VESSEL IN THE SUBMUCOUS LAYER WAS SEEN WITH AN APPARENT CELLULAR REACTION AROUND IT OF THE SAME CELLS AS ABOVE, AND IN ADDITION LYMPHOCYTES. ITS ENDOTHELIAL LINING APPEARED NORMAL. A SECTION FROM THE LARGE BOWEL AND ONE FROM THE SMALL BOWEL OF THE SECOND CASE SHOWED A SIMILAR LOCAL CONGESTION. A FEW SMALL VESSELS SHOWED SOME CELLULAR REACTION AROUND THEM AND SOME SWELLING OF THEIR ENDOTHELIUM. IN ONE FIELD, THERE WAS A COLLECTION OF CELLS, RATHER LIKE A SOLITARY FOLLICLE, THE CELLS OF WHICH HOWEVER WERE AS DESCRIBED ABOVE, RATHER THAN LYMPHOCYTES. IN THE THIRD CASE LOCAL REACTION WAS SEEN IN THE MUCOUS MEMBRANE, THE CELLS BEING AS PREVIOUSLY DESCRIBED; DEEP TO THIS, IN THE SUBMUCOUS LAYER AN ENLARGED SOLITARY FOLLICLE WAS SEEN.

SEE MICROPHOTOGRAPH TWELVE.

IN A SECOND SECTION FROM THE SAME CASE A FEW CELLS WITH LARGE OVAL NUCLEI WERE SEEN BETWEEN THE MUSCULAR LAYERS.

LIVER.

TWO SECTIONS OF LIVER WERE AVAILABLE.
ONE SECTION SHOWED MARKED GENERAL CLOUDY SWELLING. THE KUPFER CELLS WERE GENERALLY SWOLLEN AND IN SOME CASES HAD PHAGOCYTOSED RED CELLS. IN THE LIVER SINUSES MANY CELLS WERE SEEN WITH OVAL OR ROUND NUCLEI, WITH A CLEAR RETICULAR NUCLEAR STRUCTURE AND NEUTROPHYL OR FAINTLY ACID CYTOPLASM. ERYTHROCYTES WERE ALSO SEEN. THERE WAS NO INCREASE IN THE NUMBERS OF CELLS IN THE PORTAL TRACTS. IN THE OTHER SECTION CLOUDY SWELLING WAS NOT SO MARKED. PHAGOCYTOSIS BY KUPFER CELLS WAS COMMON, AND THE SINUSES CONTAINED A FEW CELLS OF THE SAME APPEARANCE AS THE FIRST SECTION. IN A FEW AREAS SMALL FOCAL COLLECTIONS OF CELLS WERE SEEN TOWARDS THE PERIPHERY OF THE LOBULE. THE CELLS WERE OF VARIED SIZE BUT WERE MAINLY LARGE WITH OVAL OR ROUND RETICULAR NUCLEI, SOME WITH FAINTLY ACID CYTOPLASM. THE SURROUNDING LIVER CELLS SHOWED FATTY DEGENERATION. THERE WAS AN INCREASE OF CELLS IN THE PORTAL TRACTS.

SEE MICROPHOTOGRAPH FOURTEEN.

CENTRAL NERVOUS SYSTEM.

SIX SECTIONS WERE EXAMINED FROM VARIOUS PARTS OF THE BRAINS OF THE THREE CASES, CEREBELLUM, CEREBRAL CORTEX AND MID BRAIN FROM THE REGION OF THE AQUEDUCT. NO PERIVASCULAR CUFFING OR CELLULAR REACTION WAS
MICROPHOTOGRAPH NUMBER THIRTEEN.

H&E. X4-00

SECTION SHOWING CELLULAR REACTION IN MUCOUS AND SUBMUCOUS LAYERS OF THE SMALL BOWEL.

THE CELLS ARE LARGE WITH OVAL OR IRREGULAR NUCLEI.

MICROPHOTOGRAPH NUMBER FOURTEEN.

H&E. X450

SECTION OF LIVER SHOWING CELLULAR REACTION IN A PORTAL TRACT. THERE IS FATTY DEGENERATION OF THE ADJOINING LIVER CELLS AND CLOUDY SWELLING OF THE REST OF THE LIVER PARENCHYMA.
SEEN IN ANY OF THESE SECTIONS. THERE WAS CONGESTION OF THE CAPILLARIES. ONE SECTION WAS RESTAINED WITH GIEMSA. NO RICKETTSIAE WERE SEEN.

ADRENAL.

ONLY ONE SECTION OF ADRENAL TISSUE WAS AVAILABLE. THERE WAS NO OBVIOUS INJURY OF THE CELLS OF THE CORTICAL LAYER. THE VESSELS APPEARED NORMAL.

PANCREAS.

ONE SECTION OF PANCREAS WAS EXAMINED. THERE WAS NO OBVIOUS ABNORMALITY.

DISCUSSION OF THE PATHOLOGY.

THE LESIONS IN GENERAL.

THE NAKED EYE APPEARANCES IN MY OWN CASES ARE CONSISTENT WITH THE DESCRIPTIONS GIVEN OF THE MORBID ANATOMY OF TSUTSUGAMUSHI DISEASE BY FAIRLEY IN PRICE'S TEXTBOOK OF MEDICINE (80). THEY RESEMBLE THE DESCRIPTION GIVEN BY MCGOVERN (180) OF FOURTEEN AUTOPSIES ON SCRUB TYPHUS IN NEW GUINEA, AND DESHMUKH'S (69) DESCRIPTION OF THE APPEARANCES IN TWELVE AUTOPSIES ON EAST AFRICANS WHO CONTRACTED THE DISEASE IN BURMA. ALSO THE DESCRIPTIONS OF TATTERSALL AND PARRY'S FINDINGS IN ASSAM (309). THEY ARE ALSO SIMILAR TO LEWTHWAITE'S (147) DESCRIPTION OF THE RURAL TYPE OF TROPICAL TYPHUS.
LEWTHWAITE AND SAVOOR (155) ESTABLISHED THE IDENTITY OF TSUTSUGAMUSHI DISEASE AND RURAL TYPHUS IN MALAYA, AND THEY ALSO SUGGEST THAT THE DIFFERENCES BETWEEN TSUTSUGAMUSHI DISEASE AND SUMATRAN MITE FEVER ARE NO MORE THAN THOSE THAT MAY DISTINGUISH TWO STRAINS OF ANY ORGANISM. THESE APPEARANCES SUGGEST CAPILLARY INJURY IN THE VARIOUS ORGANS. THE VASCULAR LESION OF EXANTHEMATIC TYPHUS, FIRST DESCRIBED IN SKIN MACULES BY FRAENKEL (97) IN 1914 AND IN THE CENTRAL NERVOUS SYSTEM BY VON PROWAZEK (322) IN 1915, IS CONSIDERED BY WOOLBACH TODD AND PALFREY (347) TO BE PRIMARILY INJURY TO CAPILLARY ENDOTHELium WITH, IN DUE COURSE, A CELLULAR REACTION ROUND THE VESSEL AND THROMBOSIS.

HISTOLOGICAL EXAMINATION HAS NOT CONFIRMED IN ALL OUTBREAKS THE CORRECTNESS OF THE ASSUMPTION THAT THE LESION OF SCRUB TYPHUS AND TSUTSUGAMUSHI DISEASE IS PRIMARILY DUE TO CAPILLARY ENDOTHELIAL DAMAGE. SOME INVESTIGATORS SUGGEST THAT ENDOTHELIAL INJURY WHEN IT OCCURS IS A RESULT OF A PROCESS BEGINNING IN THE TISSUES AROUND THE VESSEL AND IN ITS COAT. TO THE NAKED EYE NEVERTHELESS THE END RESULTS WOULD APPEAR TO BE SOMEWHAT SIMILAR.

LEWTHWAITE'S (147) INVESTIGATION ON
MALAYAN SCRUB TYPHUS PUBLISHED IN 1936 ONLY INCLUDED HISTOPATHOLOGY OF THE BRAIN. HE CONSIDERED THE LESIONS FOUND SCANTILY IN FIVE OUT OF SEVEN BRAINS WERE ESSENTIALLY SIMILAR TO THE BRAIN LESIONS IN EXANTHEMATIC TYPHUS AND ROCKY MOUNTAIN SPOTTED FEVER. THE COMMONEST LESION ACCORDING TO LEWTHWAITE (147) WAS PERIVASCULAR PROLIFERATION, ONE OR TWO CELLS DEEP, OF NEUROGLIAL CELLS (THE 'GLIASTEN' OF SPIELMYER) ALONG THE COURSE OF THE CAPILLARIES AND ARTERIOLES THAT SHOW A SLIGHT DEGREE OF SWELLING OF THEIR ENDO- THELIAL CELLS.

KOUWENAAR (140) ON THE OTHER HAND, IN A STUDY OF 23 CASES PUBLISHED IN 1940 STRESSED THAT IN MITE-BORNE TYPHUS IN THE DUTCH EAST INDIES, THE ARTERITIS BEGAN OUTSIDE THE VESSEL.

ALLEN AND SPITZ (2) IN A COMPREHENSIVE PATHOLOGICAL STUDY OF 74 CASES OF SCRUB TYPHUS FROM NEW GUINEA PUBLISHED IN 1945 SUPPORT THIS CONCEPTION OF KOUWENAAR'S.

SETTLE, PINKERTON AND CORBETT (283) IN 1945 RECORD THE RESULTS OF INVESTIGATION OF 55 FATAL CASES FROM NEW GUINEA. THEY CONSIDER THAT AS IN EXANTHEMATIC TYPHUS AND ROCKY MOUNTAIN SPOTTED FEVER THE PATHOLOGICAL CHANGES ARE THE DIRECT RESULT OF INVASION BY RICKETTSIAE OF THE ENDOTHELIAL LINING CELLS
OF VESSELS, OR SMOOTH MUSCLES OF BLOOD VESSELS, WITH RESULTING ENDOTHELIAL SWELLING AND PROLIFERATION, CELLULAR INFILTRATION OF VESSEL WALLS, PERIVASCULAR ACCUMULATIONS OF MONONUCLEAR CELLS, AND OCCASIONALLY THROMBOSIS AND HAEOMORRHAGE.

IT MUST BE REMEMBERED THAT THE MATERIAL FROM WHICH THESE CONCLUSIONS ARE DRAWN, COME FROM SITES GEOGRAPHICALLY FAR APART, WITH POSSIBLY VARIATION IN THE STRAIN OF THE INFECTING RICKETTSIA. MEGAW (196) POINTS OUT IN HIS SUMMARY OF ALLEN AND SPITZ'S ARTICLE THAT "THE FINDINGS HOWEVER CANNOT BE REGARDED AS UNIVERSALLY APPLICABLE IN ALL RESPECTS TO EACH OF THE THREE TYPHUS FEVERS. ALL THESE ARE KNOWN TO SHOW A WIDE RANGE OF VARIABILITY IN THEIR CLINICAL ASPECT, SO THAT ALTHOUGH THE MORBID HISTOLOGY OF EACH MAY CONFORM TO A GENERAL PATTERN, CONSIDERABLE VARIATION IN THE DETAILS OF THE PATTERN CAN BE EXPECTED TO OCCUR IN THE DIFFERENT OUTBREAKS OF THE DISEASE. THE DESCRIPTIONS OF THE MORBID HISTOLOGY OF LOUSE AND MITE-BORNE TYPHUS BY VARIOUS COMPETENT OBSERVERS PROVIDE CLEAR EVIDENCE THAT THESE VARIATIONS DO OCCUR".

THERE IS SCOPE FOR FURTHER INVESTIGATION OF THE EFFECT OF SCRUB TYPHUS AND OTHER TYPHUS-LIKE FEVERS ON THE KIDNEY. IT WOULD BE OF INTEREST TO BE ABLE TO CORRELATE CLINICAL AND POST-MORTEM FINDINGS WITH BLOOD CHEMISTRY.
IT SEEMS FEASIBLE TO SUSPECT THAT DISORDERED KIDNEY FUNCTION MAY ACCOUNT FOR SOME OF THE OBSERVED DROWSINESS AND STUPOR.

THE ESCHAR.

NO WHOLLY CONVINCING REASONS ARE ADVANCED TO EXPLAIN THE PRESENCE OR ABSENCE OF A LOCAL LESION. LEWTHWAITE AND SAVOOR (155) ONLY REPORT FOURTEEN DERMAL LESIONS IN ONE HUNDRED AND EIGHTY-ONE CASES. THEY STRESS THAT IT Seldom IS MORE THAN A PAPULE, DIS-APPEARS BEFORE THE CASE IS SEEN, AND IS DIFFICULT TO SEE ON A DARK SKIN. IT IS OF COURSE SOMETIMES MORE DEFINITE AND THEY QUOTE A CASE WHERE A MARK WAS VISIBLE THREE YEARS LATER. LEWTHWAITE AND SAVOOR (153) CONSIDER THAT THE GREATER INCIDENCE OF ESCHARS IN EUROPEANS IS IN PART BOUND UP WITH THEIR GREATER SENSITIVITY TO MILD SKIN INJURY.

THERE SEEMS NO DOUBT HOWEVER THAT THIS TENDENCY FOR A DERMAL LESION TO DEVELOP VARIES IN DIFFERENT EPIDEMICS. IN AN OUTBREAK AMONGST NATIVE TROOPS IN BURMA, GURBUKSH SINGH (109) RECORDS THIRTY-EIGHT ESCHARS OUT OF ONE HUNDRED AND SEVEN CASES (35.5%). TATTERSALL (308) SAW AMONGST ONE THOUSAND CASES, ELEVEN PER CENT WITH ESCHARS. THESE WERE MAINLY NATIVE TROOPS SEEN ON THE INDIA-BURMA BORDER. SANGSTER AND KAY (271) REPORT FORTY-ONE PER
CENT OF TWO HUNDRED AND THIRTY-FIVE CASES AMONGST AUSTRALIANS IN NEW GUINEA. KLEIN (138) SAW THIRTY-FOUR PER CENT IN FORTY-ONE BRITISH CASES IN THE CHIN HILLS, BURMA. AGRESS AND EVANS (1) IN SEVENTY-NINE CHINESE AND SEVEN AMERICAN CASES, SAW SEVENTY-FOUR PER CENT WITH ESCHARS. GRIFFITHS (107) IN THE DUTCH NEW GUINEA SAID NINETY-TWO PERCENT HAD ESCHARS. MENDELL (200) REPORTED SIXTY-EIGHT PER CENT OF SEVENTY-FIVE CASES AND BROWNING, RAPHAEL, KLEIN AND COBLENZ (37) EIGHTY PER CENT OF ONE HUNDRED AND SEVENTY-THREE CASES FROM NEW GUINEA.

IN CALCUTTA I SAW NINETY-TWO CASES WITH PREDOMINANTLY OXK AGGLUTININS. IN NO SINGLE CASE WAS I ABLE TO FIND ANYTHING LIKE A DERMAL LESION. THIS ALSO WAS LOW'S (163) EXPERIENCE WITH TEN CASES IN CALCUTTA.

LEWTHWAITE AND SAVOOR (149) WERE ABLE TO REPRODUCE TSUTSUGAMUSHI DISEASE IN MONKEYS WITH AND WITHOUT A DERMAL LESION, AND RURAL TYPHUS ALSO, WITH AND WITHOUT A DERMAL LESION. THE DIFFERENTIAL DIAGNOSIS OF THESE DISEASES IN THE ORIGINAL CASES FROM WHICH EACH VIRUS WAS OBTAINED, WAS THE PRESENCE OR ABSENCE OF AN ESCHAR.

ALLEN AND SPITZ (2) DISCUSS THE PROBLEM. APPARENTLY INTRACUTANEOUS INJECTIONS OF RICKETTSIA IN TSUTSGUMAGUSHI
DISEASE AND FIEVRE BOUTONNEUSE CAN PRODUCE A GROSSLY TYPICAL ESCHAR; SUBCUTANEOUS OR INTRAMUSCULAR INJECTION DOES NOT, ALTHOUGH GENERALISED INFECTION Follows. THEY SUGGEST THAT THE PROBOSCIS OF THE LARVAL TROMBICULA IN ANY CASE IN ITS ENTIRETY IS HARDLY SUFFICIENT TO PENE TRATE THE EPIDERMIS. GRANTED INFECTION IS THROUGH THE SKIN, ALTERNATIVE EXPLANATIONS ARE THAT THE RICKETTSIAE ARE INTRODUCED DIRECTLY INTO LYMPHATIC VESSELS, A MANIPULATION AS THEY SAY DIFFICULT TO CONCEIVE, OR THAT THERE IS A CERTAIN LOCAL CUTANEOUS IMMUNITY IN CERTAIN GROUPS OF PEOPLE.

LYMPH GLANDS.

A very variable finding in scrub typhus is lymphadenitis.

In Tattersall's (308) 1000 cases 92% had lymphadenitis; 82% of Gurbuksh Singh's (109) 107 cases had lymphadenitis with or without tenderness. Desmukh (69) found only 32% of 200 cases in African troops on the Burma front with generalised lymphadenitis. Enlarged mesenteric glands were found at autopsy. Klein (138) in 41 British soldiers in Burma found enlarged glands in every case and enlarged intrathoracic glands at autopsy. In Wilson's (340) case from Calcutta which was treated in Burma, glandular enlargement is not mentioned. One may presume that there was no adenitis.

Kapila and Maitra (129) from North Burma reported one case with cervical, axillary and inguinal lymphadenitis. Thus in most cases from the jungle areas lymphadenitis is present.

In Calcutta in 1943 in cases of what was serologically the same disease, lymphadenitis was in Indians practically always absent. Yet in an outbreak amongst British troops in Calcutta in 1942 adenitis was common and marked (personal communication, Lt. Col. C. M. Seward, R.A.M.C., 47 B.G.H. Calcutta).
BOYD (28) found in his OXK group which he considered the most clear cut of the serological groups in India, that adenitis was seen in only two cases out of thirty-five.

The absence of clinical lymphadenitis in Low's (163), Bardan's (16) and my own cases (165) suggest that Indians, in India, do not develop gross adenitis. In Boyd's (28) collection of 35 cases of serological OXK typhus, of which only two were noted to have glandular enlargement, 21 were British troops and 14 were Indian troops. In my own cases the histological findings of sinus catarrh and erythrophagia, seen in the 3 cases examined, support the conclusion that the glandular enlargement is a sinus reticulosis secondary to the rickettsial infection. It is obvious, of course, that the response may possibly be varied by the quantitative degree of infection, the strain of the infecting rickettsia, and the individual defence. I did not see any areas of necrosis in my sections. Allen and Spitz (2) observed necrosis in lymphoid tissue in 38% of 47 cases which they examined. Kouwenaar (140) confirms this. It is one of the observations on which Allen and Spitz base their suggestion that some of the effects of infection with scrub typhus are "hyperergic". They define
HYPEREGY AS "AN ALTERED TISSUE REACTIVITY, ONE PHASE OF ALLERGY". IT IS MANIFEST THAT SUCH A SUGGESTION REQUIRES CONSIDERABLE INVESTIGATION AND CONSIDERATION BEFORE ANY WEIGHT CAN BE ATTACHED TO IT.

THE SPLEEN.

THE PROBLEM OF HOW MUCH OF THE SPLENIC ENLARGEMENT IN TYPHUS IS DUE TO MALARIA IS SOMETIMES DIFFICULT TO ASSESS. SO OFTEN THE POSSIBILITY OF COINCIDENT MALARIA, ACUTE OR CHRONIC, COMPlicates THE QUESTION. LEWTHWAITE AND SAVOOR (155) AND KOUWENAAR (140) CONSIDER THAT THE SPLEEN IS ENLARGED IN SCRUB TYPHUS, AND THAT IS THE OPINION OF MOST CLINICIANS. IN ONE OUT OF SIXTEEN OF MY AUTOPSIES IN CALCUTTA WHAT WAS CONSIDERED TO BE MALARIAL PIGMENT WAS SEEN IN THE SPLENIC SMEAR. THE OTHER CASES WERE NEGATIVE. THE HISTOLOGICAL APPEARANCES IN THE THREE SECTIONS OF CONGESTION AND INCREASED PHAGOCYTIC ACTIVITY SUGGESTS A RESPONSE TO INFECTION. IT IS LIKELY THAT AT SOME PERIOD IN THEIR LIVES THEY WERE EXPOSED TO MALARIAL INFECTION. THE LARGE FIRM SPLEENS ARE UNLIKELY TO BE THE RESULT OF AN ACUTE INFECTION WITH TYPHUS, AND ARE CONSISTENT WITH OLD STANDING MALARIA.

THE NEGATIVE BLOOD FINDING DURING LIFE AND NEGATIVE SMEARS FROM SPLEENS AT AUTOPSY WOULD RULE OUT RECENT MALARIA. THE PIGMENT
SEEN IN SPLENIC SECTIONS, AS IN OTHER SECTIONS, WAS CONSIDERED TO BE DUE TO FIXATION METHODS. I CONSIDER IT IMPOSSIBLE TO REACH A DEFINITE CONCLUSION ON THE REACTION OF THE SPLEEN TO TYPHUS FROM THESE CASES.

THE LUNGS.

ALLEN AND SPITZ (2) FOUND INTERSTITIAL PNEUMONITIS OF A MARKED DEGREE TO BE COMMON IN SCRUB TYPHUS, IN CONTRAST WITH EXANTHEMATIC TYPHUS AND ROCKY MOUNTAIN SPOTTED FEVER. PHYSICAL SIGNS SUGGESTING PNEUMONITIS IS A VERY CONSTANT FINDING IN ALL DESCRIPTIONS OF THE DISEASE, AS IT WAS IN MY OWN OXK CASES.

ALLEN AND SPITZ'S (2) DESCRIPTIONS AND MICROPHOTOGRA PHS OF LUNG SECTIONS CLOSELY RESEMBLE THE APPEARANCES IN MY SECTIONS OF LUNGS. THAT IS, A MONONUCLEAR ALVEOLAR EXUDATE WITH A SIMILAR CELLULAR REACTION IN THE INTERSTITIAL TISSUES WITH OCCASIONAL RARE ERYTHROPHAGOCYTOSIS AND CYTOPHAGOCYTOSIS BY ALVEOLAR MONONUCLEAR CELLS. THE ONLY DIFFERENCE IS THEIR FINDING FREQUENTLY A BRONCHIOLITIS. KNEELAND AND SINETANA'S (139) DESCRIPTIONS AND MICROPHOTOGRA PHS OF A BRONCHO-PNEUMONIA OF UNDETERMINED ETIOLOGY ALSO CLOSELY RESEMBLE MY SECTIONS OF LUNG. FINDLAY'S (94) DESCRIPTIONS AND MICROPHOTOGRA PHS OF THE LUNGS OF MICE EXPERIMENTALLY
INOCULATED WITH 'Q' FEVER VIRUS ARE ALSO SIMILAR. ALLEN AND SPITZ (2) PRINT A MICROPHOTOGRAPH FROM ONE OF PINKERTON'S CASES OF TOXOPLASMOSIS WHICH SHOWS INTERSTITIAL PNEUMONITIS AND ENLARGEMENT OF THE ALVEOLAR LINING CELLS OF VERY MUCH THE SAME CHARACTER AS AN ADJOINING MICROPHOTOGRAPH FROM A CASE OF SCRUB TYPHUS. PINKERTON AND WEINMAN'S (251) REPORT OF THE FINDINGS IN A CASE OF TOXOPLASMOSIS IN MAN, REFERS TO SUBLPLEURAL HAEMORRHAGES BUT NOT TO A PNEUMONITIS. THE PULMONARY REACTION DESCRIBED BY NEUBUERGER, GEEVER AND RUTLEDGE (217) IN RHEUMATIC CARDITIS WAS ALSO CONSIDERED BY ALLEN AND SPITZ TO BE ESSENTIALLY SIMILAR. KOUWENAAR (140) ALSO DRAWS ATTENTION TO THE INTERSTITIAL REACTION IN THE LUNG TISSUES, AND CONSIDERS THAT IT IS DUE TO PERIVASCULAR INFILTRATION.

WHATEVER THE MECHANISM OF THE PRODUCTION OF THE RICKETTSIAL LESIONS IN THE LUNGS I HAVE NO DOUBT THAT THESE LESIONS MUST BE AN IMPORTANT CONTRIBUTING FACTOR TOWARDS A FATAL ISSUE.

THE KIDNEYS.

THE APPEARANCES OF THREE SECTIONS FROM MY THREE CASES WERE VARIABLE. INTERSTITIAL REACTIONS WERE PRESENT IN TWO SECTIONS SUBSTANTIALLY AS DESCRIBED BY ALLEN AND SPITZ (2)
AND KOUWENAAR (140). THE GLOMERULAR CHANGES DESCRIBED AND ILLUSTRATED BY ALLEN AND SPITZ (2) ARE NOT SO APPARENT. IN ONE SECTION THE GLOMERULI DID APPEAR TO BE SWOLLEN, YET THERE WAS NO ISCHAEMIA OF THE GLOMERULAE. I WAS NOT CONVINCED THAT IT WAS A TRUE GLOMERULO-NEPHRITIS.

THE FOCAL CORTICAL LESIONS SEEN IN ONE SECTION, IN APPEARANCE SEEM CONSISTENT WITH THE LESIONS IN OTHER ORGANS AND WOULD APPEAR TO BE A TYPHUS EFFECT. FURTHER INVESTIGATION IS NEEDED TO DECIDE WHETHER ALLEN AND SPITZ'S SUGGESTION THAT WHAT THEY SAW IN 30% OF THEIR SCRUB TYPHUS CASES AND 60% OF THEIR EPIDEMIC TYPHUS CASES IS REALLY ACUTE GLOMERULO-NEPHRITIS. THEY DISCUSS THE POSSIBILITY OF IT BEING EITHER A "HYPERERGIC" EFFECT OR DUE TO TOXAEMIA. THEY INCLINE TO IT BEING "HYPERERGIC" THOUGH THEY DO POINT OUT THAT THEIR CONCLUSION THAT THE APPEARANCE IS DUE TO A GLOMERULO-NEPHRITIS MAY NOT BE ACCEPTED BY ALL PATHOLOGISTS. IT SEEMS PECULIAR THAT THEY SHOULD FIND THE SAME CONDITION IN 60% OF 24 CASES OF EPIDEMIC (EXANTHEMATIC) TYPHUS, WHERE SO MANY COMPETENT PATHOLOGISTS HAVE FAILED TO REPORT IT. IF THE "HYPERERGIC" EXPLANATION IS TO BE CONSIDERED ANALAGOUS TO THE ALLERGIC HYPOTHESIS OF "ESSENTIAL" ACUTE GLOMERULO-NEPHRITIS AND
RHEUMATIC FEVER, THEN IT DIFFERS FROM THESE TWO CONDITIONS IN THAT THE "HYPERERGIC" ELEMENT INTRODUCES ITSELF AT THE HEIGHT OF WHAT MAY BE PRESUMED TO BE THE PRIMARY INFECTION. McGOVERN (180) Seldom found changes in the kidney, nor do settle, PINKERTON AND CORBETT (283). ALLEN AND SPITZ (2) IN THEIR DISCUSSION ON THE KIDNEY CHANGES NOTE THAT KAWAMURA, IN A COMPREHENSIVE DISCUSSION DID NOT MENTION GLOMERULAR DAMAGE. KOUWENAAR (140) STATES THAT "FOR THE MOST PART THE GLOMERULI ARE INTACT; OCCASIONALLY THERE IS DEFINITE CAPSULAR ADHESION".

SEE MICROPHOTOGRAPHS FIVE AND SEVEN.

IN EPIDEMIC TYPHUS, WOOLBACH, TODD AND PALFREY (347?) NOTE GLOMERULO-NEPHRITIS IN ONE OF THIRTY-SEVEN CASES AND INTRA-CAPILLARY PROLIFERATION IN NINE OTHERS. CEELIN (46) AND MUNK (207) FOUND NO GLOMERULAR ALTERATION. IN ROCKY MOUNTAIN SPOTTED FEVER WOOLBACH (345) AND LILLE (158) FOUND ESSENTIALLY NORMAL GLOMERULI EXCEPT IN ONE OF WOOLBACH'S FIVE CASES. LILLE (158) NOTES HOWEVER THAT PERIVASCULAR SMALL CELLED INFILTRATION HAS BEEN VERY FREQUENTLY SEEN IN TYPHUS.

ALLEN AND SPITZ (2) NOTE THAT IN EPIDEMIC TYPHUS CAFFARENA (42), FOUND DIFFUSE GLOMERULO-NEPHRITIS IN 67.5 PER CENT OF HIS
SCHOFFER (278) suspected early glomerulo-nephritis in his cases; DAWYDOWSKIE (65) saw focal destructive glomerulitis in one-third of his cases and WETZEL (331) described a case of glomerulo-nephritis in epidemic typhus. ALLEN AND SPITZ (2) consider their interpretations of the glomerular changes in their material approximate to the conclusions of CAFFARENA (42). They also consider that these changes are the remote effects of the Rickettsiae, and that this is essentially also the view of JULLIARD AND HENAFF (128). ANDREW (4) refers to a sudden diuresis with disappearance of oedema on the tenth to the fourteenth day in ten or of eleven cases. This he considered to be a disturbance of the fluid balance unconnected with the kidney. There was no sign in the urine of active nephritis and there was a striking improvement in the condition of the patients coincidental with the diuresis. RENARD AND RAYNAUD (262) refer to a case of exanthematic typhus with nephritis, where recovery followed decapsulation of the kidney. They considered the nephritis was due to the typhus, and that it is common in many cases of typhus. Such a degree of oedema has not been described in scrub typhus nor was it apparent in any naked eye appearances in my autopsies.
SNYDER, MURRAY, ECKE AND ZARAFONETIS (350) report that the severest cases of epidemic typhus seen in 64 Egyptians were in a state of azotaemia - when the non protein nitrogen was found by them to be 45 mg.% or over. Granular casts were rare however. Erythrocytes and white cells were often seen in the urinary sediment. Megaw (197) in his abstract of this article by Yeomans and his collaborators considers that these patients may be exceptional owing to their poor state, low protein intake, and the virulence of the strain of typhus. Dehydration was admitted to an important contributing factor.

I consider that the effect of the disease on the kidney requires further investigation. From the literature it would seem that there may be considerable variation in the degree of kidney damage. I cannot in my own series produce any results of precise investigations of kidney function. I consider that the histological lesions seen in the three cases studied might very well have been a factor contributing to the fatal issue.

The heart.

Settle, Pinkerton and Corbett (283) considered, that myocarditis was a cause of death in 50% of their fifty-five cases. Allen
AND SPITZ (2) FOUND VARYING DEGREES OF MYOCARDITIS IN 93% OF 74 CASES. McGOVERN (180) FOUND MYOCARDITIS, WHICH APPEARED TO BE SECONDARY TO VASCULITIS. MENDELL (200) IN TWO AUTOPSIES ONLY, NOTED THE VASCULITIS AND PERIVASCULITIS IN THE SMALLER VESSELS OF THE HEART. KLEIN (138) FOUND MONONUCLEAR INFILTRATION BETWEEN THE MUSCLE FIBRES OF THE HEART IN 5 AUTOPSIES. AS I HAD ONLY SECTIONS FROM TWO OF MY CASES AVAILABLE FOR EXAMINATION, IN ONE OF WHICH THERE WAS PROBABLY AN ANTICEDENT RHEUMATIC INFECTION, I AM UNABLE TO SAY WHETHER MYOCARDITIS WAS COMMONLY PRESENT OR NOT. IN VIEW OF THE FINDINGS ELSEWHERE ONE WOULD EXPECT THAT THE HEART TO BE INVOLVED IN A PERCENTAGE OF CASES.

THE GASTRO-INTESTINAL TRACT.

THE FINDINGS IN THE BOWEL WERE MICROSCOPICALLY SIMILAR TO THE REACTIONS IN OTHER TISSUES. MICROSCOPICALLY THEY RESEMBLE GENERALLY THE FINDINGS OF OTHER INVESTIGATORS, KOEKENAAR (140), McGOVERN (180) AND DESMUKH (69). LEWTHWAITE (147) ONLY FOUND HAEMORRHAGES WITHIN THE MUCOSA OF THE STOMACH IN TWO CASES. IT IS OF INTEREST TO NOTE THE LACK OF GENERAL MICROSCOPIC ENLARGEMENT OF LYMPHOID FOLLICLES. LEWTHWAITE (147) RECORDS IT IN ONE OF HIS TWELVE CASES. THE LOCAL LYMPHOID HYPERPLASIA SEEN MICROSCOPICALLY MAY BE PRODUCED BY A LOCAL LESION.
I have seen localized congestion of the mucous membrane at various points along the alimentary tract, in cases of enteritis due to *B. paratyphoid C* and *C*. and *B. enteritidis* Gaertner, where the lesions are presumably due to toxic action on the capillaries. There is no reason, however, why some of these lesions seen in scrub typhus should not be due to the presence of rickettsiae in the tissues, which further search would reveal.

**The Liver.**

The liver changes in the only two sections available were consistent with some of the changes seen by Allen and Spitz (2) namely the swelling of the Kupffer cells with erythrophagocytosis and cytophagocytosis, the increase of cells in the sinusoids, the scattered foci of cells with fatty degeneration of surrounding liver cells and the slightly increased concentration of mononuclear cells in the portal tracts.

**The Central Nervous System.**

The difficulty in finding lesions in the central nervous system in scrub typhus is well known. Lewthwaite (147) in a prolonged search failed to find lesions in two of seven cases. In the other cases only a few lesions were seen. It is therefore not surprising that I was unable to find a suspicious
APPEARANCE IN ANY OF SIX SECTIONS FROM THREE CASES. ALLEN AND SPITZ (2) WERE ABLE TO DEMONSTRATE LESIONS IN 30% OF THEIR CASES, AND SPECIFIC LEPTOMENINGITIS IN 89% OF CASES. KOUWENAAR (140) WAS ALSO ABLE TO DEMONSTRATE LESIONS.

I AM NOT CONVINCED THAT THESE LESIONS, IN SUCH SMALL NUMBERS, OF THEMSELVES PRODUCE THE CLINICAL DROWSINESS AND SLOW CEREBRATION. THIS EFFECT IS PRODUCED IN SOME OTHER WAY, PERHAPS BY OEDEMA, OR TOXAEMIA, OR TO THE EFFECTS OF THE LESIONS IN THE KIDNEY WITH DISTURBANCE OF ITS FUNCTION.

THE ADRENAL.

THE APPEARANCE OF ONE SECTION DID NOT SUPPORT ALLEN AND SPITZ'S (2) SUGGESTION THAT ADRENAL CORTICAL DAMAGE WAS A Factor IN THE DISEASE.

SUMMARY OF THE PATHOLOGY.

IT SEEMS TO ME THAT THE FATAL EFFECTS OF SCRUB TYPHUS INFECTION CANNOT AS YET BE EXPLAINED READILY AND SIMPLY. IT IS TRUE THAT FROM CASE TO CASE, LESIONS, AND EXTENSIVE LESIONS ARE PRESENT IN VARYING ORGANS. AS I HAVE MENTIONED THE CENTRAL NERVOUS SYSTEM LESIONS IN SCRUB TYPHUS ARE EXCEEDINGLY SCANTY. AFTER WITNESSING THE LACK OF CORRELATION BETWEEN THE CLINICAL STUPOR AND THE
HISTOLOGICAL APPEARANCES I AM NOT IMPRESSED WITH STRUMM'S (305) ATTEMPT TO EXPLAIN ALL THE EFFECTS OF EXANTHEMATIC TYPHUS, WHERE ADMITTEDLY THE NERVOUS LESIONS ARE MORE NUMEROUS, BY A CENTRAL ACTION OF THE NATURE OF "DESTRUCTIVE PROLIFERATIVE THROMBO-ANGEITIS INTERFERING WITH THE BLOOD SUPPLY OF VARIOUS CENTRES ESPECIALLY THOSE OF THE HYPOTHALMUS". THESE EFFECTS WHICH HE MENTIONS, RANGE FROM TACHYCARDIA AND LOW BLOOD PRESSURE TO NEPHRITIS, HYPRETHYROIDISM AND HYPOCALCAEMIA. THE LUNGS ARE COMMONLY INVOLVED AND OFTEN EXTENSIVELY, BUT SOMETIMES QUITE A REASONABLE AMOUNT OF NORMAL LUNG TISSUE WOULD APPEAR TO BE FUNCTIONING. AS I HAVE SAID I CONSIDER THE LUNG LESION WHEN PRESENT, A MOST IMPORTANT CONTRIBUTORY FACTOR TOWARDS DIS-SOLUTION. THE OVERALL SIGNS OF TOXAEMIA ARE PRESENT IN LIVER AND KIDNEY. THE HEART LESIONS WHEN PRESENT MUST CONTRIBUTE THEIR QUOTA OF DISTRESS. THE LESIONS IN THE KIDNEY ARE AN OBVIOUS POSSIBLE CAUSE FOR SOME OF THE GENERAL TOXIC SYMPTOMS.

FACTUAL INFORMATION ON TYPHUS IN INDIA IS STILL EXCEEDINGLY SCANTY, AND OPINIONS ON MANY POINTS ARE FORMED BY ANALOGY FROM TYPHUS IN OTHER COUNTRIES. "TOXAEMIA" IS FREQUENTLY EVOKED AS AN IMPORTANT CAUSE OF DEATH. IT IS
NOT UNREASONABLE TO SUPPOSE THAT THE RICKETTSIAE DURING THEIR EXISTANCE PRODUCE TOXIC SUBSTANCES, BUT AS THESE SUBSTANCES ARE NOT CAPABLE OF PRECISE ESTIMATION, AND AS FAR AS I KNOW, THEIR EFFECTS HAVE NOT BEEN STUDIED IN THE SAME WAY AS BACTERIAL TOXINS, I AM A LITTLE INCLINED TO REGARD THEIR ACTION AS PERHAPS OVERATED. CONTRIBUTORY CAUSES ARE THE SECONDARY LESIONS PRODUCING ANOXAEMIA FROM PNEUMONITIS AND ON OCCASIONS A BACTERIAL PNEUMONIA, DISTURBANCES OF EXCRETION FROM INTERSTITIAL NEPHRITIS AND THE STRAIN ON THE CIRCULATION FROM INTERSTITIAL MYOCARDITIS.

DISCUSSION.

TYPHUS FEVER IS FOUND IN CERTAIN PARTS OF INDIA AND TYPHUS-LIKE FEVERS ARE FOUND ALL OVER THE CONTINENT OF INDIA, IN BURMA AND IN CEYLON. SINCE MEGAW (186) FIRST DREW ATTENTION TO THE PRESENCE OF A TYPHUS-LIKE FEVER IN THE KUMAON, CASES HAVE BEEN REPORTED IN INCREASING NUMBERS FROM AREAS IN WHICH THE DISEASE WAS NOT THOUGHT TO OCCUR. THIS APPARENT INCREASE WAS PROBABLY DUE TO IMPROVEMENT IN DIAGNOSIS AND INCREASED AWARENESS OF THE CLINICIAN OF ITS PRESENCE. UNTIL THE OUTBREAK OF THE SECOND WORLD WAR, CASES WERE SPORADIC OR IN SMALL OUTBREAKS. THE EPIDEMICS OF SCRUB TYPHUS (TSUTSUGAMUSHI
DISEASE) DURING THE CAMPAIGNING IN ASSAM AND BURMA MADE IT EVIDENT THAT THE DISEASE WAS ENDEMIC IN THOSE AREAS. THE WORK OF MACKIE (169) AND HIS CO-WORKERS HAS IDENTIFIED THE ANIMAL HOSTS AND VECTORS CONCERNED. THEY HAVE ISOLATED NUMEROUS STRAINS OF THE CAusal Rickettsia. WHAT ARE APPARENTLY SIMILAR STRAINS OF Rickettsia HAVE BEEN ISOLATED BY PARKER AND SAVOOR (239 & 240) FROM CASES NEAR IMPHAL IN ASSAM AND FROM A CASE IN CALCUTTA. THOUGH THE HOSTS AND VECTORS HAVE NOT BEEN ESTABLISHED IN PARKER AND SAVOOR'S CASES, ON ANALOGY IT IS PROBABLE THAT THESE ARE SIMILAR TO THE HOSTS AND VECTORS IN ASSAM AND ELSEWHERE. THE PROBABLE CAUSE OF THESE OUTBREAKS WOULD SEEM TO BE THE CLOSE CONTACT MADE BY TROOPS WITH THE JUNGLE, AND THE DISTURBANCE OF THE NATURAL VECTOR HOST CIRCLE. THIS HAS BEEN NOTED ELSEWHERE BY HAY (113).

WEBSTER (326) IN 1940 REPORTED THE ISOLATION IN SIMLA OF STRAINS OF SeroLOGIcALLY OXK. HE HAS BEEN UNABLE HOWEVER TO ISOLATE A STRAIN FROM A POSSIBLE VECTOR.

MURINE STRAINS HAVE BEEN ISOLATED IN SIMLA FROM RATS AND RAT FLEAS BY COVELL (54) AND COVELL AND MEHTA (55).
WOLFF (342) has described scrotal changes in guinea pigs injected with an emulsion of rat fleas in Colombo. Smears from the tunica vaginalis showed rickettsiae. Heilig and Naidu (117 & 118) have isolated rickettsiae from cases in South India. These cases, on the basis of complement fixation tests would seem to be related to rocky mountain spotted fever.

Clinically the cases resemble mild Rocky Mountain spotted fever. No tick in India has ever been found to be infected with rickettsiae. Factual information is particularly scanty about this type of case of the indeterminate Weil-Felix group. Facilities for investigation are poor in India. Many of the conclusions regarding the vector and the type of disease are based on clinical observations, and on analogy with typhus-like fevers seen elsewhere.

No outbreaks of the indeterminate group of typhus-like fever have been seen comparable to the outbreaks of Tsutsugamushi fever (scrub typhus) seen in Assam, Burma and Ceylon during the recent war.

One may reasonably conclude however that in India and possibly Burma, cases representative of the three main groups of typhus are found.

One cannot but compare the Indian
SUB-CONTINENT WITH THE CONTINENT OF AFRICA. IN AFRICA CASES REPRESENTING THE EXANTHEMATIC-MURINE TYPHUS GROUP AND THE ROCKY MOUNTAIN SPOTTED FEVER-FIEVRE BOUTONNEUSE GROUP HAVE BEEN REPORTED. NO CASES RESEMBLING TSUTSUGAMUSHI FEVER HAVE BEEN SEEN. IT IS THE OPINION OF THE SOUTH AFRICAN WORKERS PIJPERS AND DAU (247) THAT THE SOUTH AFRICAN CASES DO NOT EXACTLY RESEMBLE THEIR PROTOTYPES ELSEWHERE. FELIX (86) GOES SO FAR AS TO PLACE ALL SOUTH AFRICAN TYPHUSES IN ONE SEROLOGICAL GROUP, THE INDETERMINATE GROUP WHICH INCLUDES ROCKY MOUNTAIN SPOTTED FEVER AND FIEVRE BOUTONNEUSE.

THE VECTOR, HOST AND MODE OF INFECTION FOR TSUTSUGAMUSHI FEVER (SCRUB TYPHUS) SEEMED, FROM MACKIES (169) INVESTIGATIONS, TO BE ESSENTIALLY SIMILAR IN ASSAM AND BURMA TO THE MODE OF INFECTION IN OTHER COUNTRIES, WHERE THE DISEASE IS PRESENT. BY ANALOGY, IN THOSE CASES OF THE INDETERMINATE GROUP, A TICK VECTOR MIGHT REASONABLY BE SUSPECTED. SO FAR, HOWEVER, THE INVESTIGATIONS OF MEGAW (187, 189, 190, 191 & 192), BLEWITT (26), CHRISTIAN (49), STOTT (303), HEILIG (116) AND HEILIG AND NAIDU (117, 118 & 119) HAVE BEEN UNABLE TO IMPLICATE THE TICK. LIKEWISE INVESTIGATIONS HAVE BEEN NEGATIVE IN A SEARCH FOR A POSSIBLE HOST FOR THIS TYPE.

THE LOUSE BY ANALOGY MIGHT BE
INVESTIGATED IN POSSIBLE CASES OF EXANTHEMATIC TYPHUS. SO FAR THIS HAS NOT BEEN DONE, NOR HAS A STRAIN BEEN ISOLATED FROM A CASE. NOR HAS MURINE TYPHUS BEEN IDENTIFIED WITH CERTAINTY. ON CLINICAL AND SEROLOGICAL GROUNDS HOWEVER IT MAY BE ASSUMED THAT EXANTHEMATIC AND MURINE TYPHUS IS PRESENT.

AT PRESENT FOR PRACTICAL PURPOSES THE ONLY LABORATORY AID TO DIAGNOSIS IN INDIA IS THE WEIL-FELIX TEST. THIS TEST IS OF GREAT VALUE IF INTERPRETED WITH ITS RECOGNISED LIMITATIONS. DICK'S (70) OBSERVATIONS IN THE MIDDLE EAST OF NON SPECIFIC REACTIONS HAVE BEEN PRONOUNCED BY SUCH AN AUTHORITY AS FELIX (89) TO HAVE BEEN PROBABLY CAUSED BY DETERIORATION OF SUSPENSIONS.

RAISED TITRES ARE ENCOUNTERED HOWEVER IN BRUCELLIASIS, TOXOPLASMOSIS AND SUPPURATIVE PROCESSES, PARTICULARLY DUE TO BACILLUS PROTEUS. BORDERLINE DIAGNOSTIC TITRES MAY BE FOUND IN TYPHOID FEVER, PARATYPHOID A, B AND C, SALMONELLA ENTERITIDIS GAERTNER INFECTIONS AND POSSIBLY ALSO IN KALA AZAR. CLINICALLY THE DIAGNOSIS MAY BE EXCEEDINGLY DIFFICULT, ESPECIALLY IN SPORADIC CASES AND PARTICULARLY IN THE ABSENCE OF A RASH. THE RASH IF PRESENT IS HELPFUL. IN TSUTSUGAMUSHI FEVER IT IS OFTEN NOT AT ALL EASY TO DETECT, PARTICULARLY ON THE
SKINS OF NATIVES OF INDIA. THE RASHES OF THE CASES DESCRIBED BY HEILIG AND NAIDU (117 & 118) ARE MUCH MORE PROMINENT AND WERE SEEN IN ALL CASES. THE CENTRIFUGAL ONSET OF THESE RASHES RESEMBLES ROCKY MOUNTAIN SPOTTED FEVER. THE LOCUS OF INFECTION AND THE SEASON OF THE YEAR MAY BE SUGGESTIVE. WITHOUT LABORATORY FACILITIES HOWEVER, THE DIAGNOSIS MUST IN MANY CASES REMAIN IN DOUBT. SERIAL WEIL-FELIX TESTS ARE OFTEN ESSENTIAL TO BRING OUT A WAXING AND WANING TITRE. AN ESCHAR IS COMMON IN EUROPEAN CASES OF TSUTSUGAMUSHI FEVER AND LESS COMMON IN NATIVE CASES.

THE INDIAN LITERATURE CONSISTS MAINLY OF CLINICAL REPORTS OF ISOLATED CASES OR SERIES OF CASES. LANDMARKS ARE MEGAW'S (186) DESCRIPTION OF HIS OWN CASE IN 1917 AND HIS REFERENCE TO McKECHNIE'S (181) UNPUBLISHED REPORT FROM THE SAME AREA IN 1913. IN 1924 PHIPSON (245) REPORTED THE FIRST CASES DIAGNOSED IN THE ABSENCE OF A RASH BY MEANS OF THE WEIL-FELIX TEST. BOYD (28) IN 1935 PUBLISHED THE RESULTS OF A STUDY OF 108 CASES REPORTED IN THE ARMY IN INDIA IN 1934, IN WHICH STANDARDISED PROTEUS SUSPENSIONS WERE USED. THIS WAS UP TO DATE THE ONLY ATTEMPT TO CORRELATE AS FAR AS WAS POSSIBLE
THE TYPHUS-LIKE FEVERS OF INDIA USING A STANDARDISED WEIL-FELIX TECHNIQUE. BLEWITT (26) IN 1936 MADE THE FIRST FIELD INVESTIGATION, WITH SOMEWHAT INCONCLUSIVE RESULTS IN THE KUMAON AREA, WHERE MEGAW CONTRACTED THE DISEASE. COVELL AND HIS COLLABORATORS (54, 55, 56 & 57) IN 1936 AND WEBSTER (326) IN 1940 REPORTED THE ISOLATION OF RICKETTSIAE IN MAN AND IN POSSIBLE HOSTS AND VECTORS IN SIMLA. MAITRA AND SEN GUPTA (173) IN BURMA IN 1936 REPORTED 109 POSITIVE WEIL-FELIX RESULTS IN SERA SENT FOR WIDAL TESTS. SIMILARLY WOODHEAD AND DUTTA (343) IN 1941 IN ASSAM REPORTED 203 POSITIVE WEIL-FELIX RESULTS IN WIDAL SERA. SCRUB TYPHUS AND MURINE TYPHUS HAVE BEEN REPORTED FROM CEYLON BY NICHOLS (218) AND WIJERAMA (334). HEILIG AND NAIDU (117, 118 & 119) IN 1941, 1942 AND 1944 PUBLISHED THEIR IMPORTANT PAPERS DESCRIBING THEIR CASES, AND IN COLLABORATION WITH TOPPING (119) REPORTING THE RELATIONSHIP THROUGH THE COMPLIMENT FIXATION TEST WITH ROCKY MOUNTAIN SPOTTED FEVER. BARĐAN (16) IN 1944 REPORTED CASES WITHOUT A RASH AND WITH SYMPTOMS WITH A "CLOSE RESEMBLANCE TO ANY ACUTE UPPER RESPIRATORY INFECTION OF INFLUENZAL TYPE". IN 1946, MACKIE (169) AND HIS COLLABORATORS PUBLISHED THE RESULTS OF THE REALLY THOROUGH FIELD INVESTIGATION BACKED BY
ADEQUATE LABORATORY WORK.

THE GENERAL DISTRIBUTION OF THE VARIOUS TYPHUS-LIKE FEVERS IN INDIA CAN AT PRESENT ONLY BE OUTLINED VAGUELY. EXANTHEMATIC LOUSE-BORNE TYPHUS IS REPORTED FROM THE NORTHERN FRONTIERS. THE HABITS OF THE HILL PEOPLE ARE CONSISTENT WITH LOUSE SPREAD DISEASE. BALBIR SINGH (13) HAS REPORTED A POSSIBLE OUTBREAK IN "PAI FORCE" OF EXANTHEMATIC TYPHUS THOUGHT TO HAVE BEEN SPREAD BY REFUGEES FROM THE BALTIC AND THE UKRAINE.

MURINE TYPHUS HAS BEEN FOUND IN SIMLA AND ON SEROLOGICAL RESULTS IS PROBABLY PRESENT IN BOMBAY, BANGALORE, CALCUTTA, RANGOON, COLOMBO AND OTHER LARGER TOWNS. TSUTSUGAMUSHI FEVER IS ENDEMIC IN VARIOUS AREAS IN UPPER BURMA, ASSAM, BENGAL AND AROUND POONA IN SOUTH INDIA. IT IS PROBABLY PRESENT IN JUNGLE AREAS ELSEWHERE IN INDIA AND CEYLON. CASES HAVE BEEN SEEN IN WHICH THE INFECTION WAS APPARENTLY CONTRACTED IN GREATER CALCUTTA. THE TYPHUS-LIKE FEVER OF INDETERMINATE SEROLOGICAL GROUP IS FOUND IN MYSORE IN SOUTH INDIA AND IN VARIOUS AREAS ALL OVER CENTRAL INDIA.

DURING THE 1939-45 WAR, WHILE IN AN INDIAN MILITARY HOSPITAL IN CALCUTTA, I SAW ONE HUNDRED AND FOURTEEN CASES OF TYPHUS-LIKE FEVER. SIXTY OF THESE CASES WERE SENT FROM A BRIGADE IN TRAINING NEAR JESSORE, EIGHTY MILES
TO THE NORTH. THE REST WERE FROM THE GREATER CALCUTTA AREA.

OF SOME OF THE PATIENTS. THREE OF THESE CASES AGGLUTINATED OXK ALONE, TWO AGGLUTINATED OXK WITH CROSS-AGGLUTINATION FOR OX19, ONE CASE AGGLUTINATED OX19 ONLY AND ONE CASE AGGLUTINATED OX2 WITH CROSS-AGGLUTINATION FOR OX19.

AN OXK STRAIN OF RICKETTSIA WAS ISOLATED BY PARKER AND SAVOOR (239 & 240) ON THE TWELFTH DAY FROM A CALCUTTA CASE ULTIMATELY FATAL ON THE SIXTEENTH DAY. SMADEL, RIGHTS AND JACKSON (288) FOUND THIS STRAIN INDISTINGUISHABLE FROM AN OXK ASSAM STRAIN, ON THE BASIS OF CROSS-IMMUNITY AND COMPLIMENT FIXATION TESTS.

IT SEEMS LIKELY THAT THESE CASES CONSISTED OF TSUTSUGAMUSHI DISEASE (SCRUB TYPHUS), MURINE TYPHUS AND THE INDETERMINATE GROUP OF TYPHUS-LIKE FEVERS.


VARIOUS LABORATORY INVESTIGATIONS WERE CARRIED OUT. THE TOTAL WHITE BLOOD CELL

THE HIGHEST TITRE OBSERVED WAS 1/16 IN ONE CASE. THESE OBSERVATIONS DO NOT SUGGEST THAT THESE AGGLUTININS WERE INCREASED IN MY CASES IN CALCUTTA. FURTHER OBSERVATIONS ARE REQUIRED BEFORE ONE COULD DRAW ANY CONCLUSIONS OF ITS POSSIBLE VALUE IN DIFFERENTIATING THE
SO CALLED ATYPICAL PNEUMONIAS FROM THE "PNEUMONITIS" OF A MILD SCRUB TYPHUS.

COMPLICATIONS WERE FEW. PAROTITIS (TWO CASES), BLEEDING GUMS (ONE CASE), HAEMORRHAGE FROM THE BOWEL (ONE CASE), HAEMATURIA (TWO CASES), HAEMOPTYSIS (THREE CASES), SEVERE EARACHE (ONE CASE), MARKED DEAFNESS (ONE CASE), AND MENTAL CONFUSION PERSISTING INTO CONVALESCENCE WITH ULTIMATE RECOVERY (TWO CASES) WERE NOTED. THE HAEMORRHAGES MAY HAVE BEEN DUE TO THE LOCAL TYPHUS LESION. IN THE TWO CLINICAL CASES WITH HAEMATURIA, LOCAL BLADDER LESIONS WERE SEEN AT AUTOPSY.

ELEVEN CASES WERE VERY MILD FORMS OF THE DISEASE WHO WERE ABLE TO WALK SLOWLY ROUND THE WARD AND CONSUME A FAIRLY LIBERAL DIET DURING THEIR ILLNESS.

SIXTEEN CASES WERE AUTOPSIED. ELEVEN CASES WERE SEROLOGICALLY OXK, WITH NO CROSS-AGGLUTININS. FIVE WERE DIAGNOSED ON CLINICAL GROUNDS AND ON PATHOLOGICAL APPEARANCES. THE NAKED EYE PATHOLOGY WAS OF THE SAME NATURE AS DESCRIBED BY MCGOVERN (180) IN NEW GUINEA, ALLEN AND SPITZ (2) IN NEW GUINEA, DESHMUKH (69) IN BURMA, TATTERSALL AND PARRY (309) IN BURMA AND LEWTHWAITE AND SAVOOR (155) IN MALAYA, AND AS DESCRIBED BY FAIRLEY (80) IN PRICE'S TEXTBOOK. AS LEWTHWAITE (147) HAS PUT
IT, THERE IS "NO SINGLE CONSISTENT FEATURE ON NAKED EYE EXAMINATION PECULIAR TO TROPICAL TYPHUS. THE GROSS FINDINGS CONSISTED RATHER OF A FEW PATHOLOGICAL CHANGES, USUALLY ONLY OF SLIGHT DEGREE, EACH OF WHICH IS TO BE OBSERVED INDIVIDUALLY AND SOME OF THEM COLLECTIVELY IN THE MORBID ANATOMY OF OTHER DISEASES".

MICROSCOPIC EXAMINATION FAILED TO DEMONSTRATE RICKETTSIAE, AND THE MORBID HISTOLOGICAL APPEARANCES WERE NOT SUGGESTIVE OF LESIONS OF VESSELS SUCH AS ONE PERHAPS EXPECTED. I WAS LATER ABLE TO STUDY SLIDES FROM THREE OXK CASES. IN THESE THREE CASES NO OBVIOUS SPECIFIC LESIONS WERE SEEN IN THE MYOCARDIUM, CENTRAL NERVOUS SYSTEM, ADRENAL OR PANCREAS. IN THE LUNGS PATCHES OF INTERSTITIAL CONGESTION WERE SEEN, WITH IN MANY ALVEOLI, A MONONUCLEAR ALVEOLAR EXUDATE WITHOUT OBVIOUS FIBRIN. THE APPEARANCE WAS SIMILAR TO DESCRIPTIONS AND MICROPHOTOGRAPHS OF KNEELAND AND SMETANA'S (139) BRONCHOPNEUMONIA OF UNDETERMINED ETIOLOGY, FINDLAY'S (94) DESCRIPTIONS AND MICROPHOTOGRAPHS OF EXPERIMENTAL 'Q' FEVER IN MICE LUNGS, THE PULMONARY REACTION DESCRIBED BY NEUBUERGER, GEEVER AND RUTLEDGE (217) IN RHEUMATIC CARDITIS AND PINKERTON'S (249) MICROPHOTOGRAPHS OF THE LUNGS IN A CASE OF TOXOPLASMOSIS MENTIONED BY ALLEN AND SPITZ. IT IS NOT
POSSIBLE TO REACH ANY CONCLUSION REGARDING THE EXACT LOCUS OF INFECTION IN THE LUNG OR ITS MECHANISM WITHOUT FURTHER INVESTIGATION. THE SIMILAR APPEARANCE IN THE LUNGS OF MICE EXPERIMENTALLY INFECTED WITH 'Q' FEVER, AND THE PROOF OF THE PRESENCE OF RICKETTSIAE IN THESE LUNGS WHEN USED TO PASSAGE THE INFECTION, SUGGESTS THAT POSSIBLY RICKETTSIAE ORIENTALIS MAY BE PRESENT IN THE LUNG TISSUE OF MY CASES, ALTHOUGH NOT OBVIOUS BY ORDINARY STAINING METHODS.

THE MORBID HISTOLOGY OF THE KIDNEY IS OF INTEREST, AS IT SEEMS LIKELY THAT AN UNSUSPECTED REACTION IN THAT ORGAN MAY BE OF IMPORTANCE IN TREATMENT. IN TWO OF MY CASES THE INTERSTITIAL CHANGES WERE SUBSTANTIALLY AS DESCRIBED BY ALLEN AND SPITZ (2) AND KOUWENAAR (140), THOUGH THE GLOMERULAR CHANGES WERE NOT SO OBVIOUS. THE FOCAL INTERSTITIAL REACTIONS AND IN OTHER SECTIONS INTERSTITIAL OEDEMA, AND THE GLOMERULAR REACTIONS IN SOME PLACES SUGGEST A CONSIDERABLE INTERFERENCE WITH KIDNEY FUNCTION. ALLEN AND SPITZ (2) ARE INCLINED TO THINK THAT THESE CHANGES ARE OF AN ALLERGIC NATURE. UNFORTUNATELY IN MY CASES NO BLOOD CHEMISTRY OR TESTS OF RENAL FUNCTION ARE AVAILABLE. THE CORRELATION OF CLINICAL FINDINGS, BLOOD CHEMISTRY AND MORBID HISTOLOGY WOULD BE OF
CONSIDERABLE INTEREST IN VIEW OF THE DROWSINESS OFTEN NOTED AND ABSENCE OF OBVIOUS MICROSCOPIC LESIONS IN THE CENTRAL NERVOUS SYSTEM.

THE ERYTHROPHAGOCYTOSIS SEEN IN SPLEEN, LUNG, LYMPHOID TISSUE AND LIVER SINUSOIDS WOULD SEEM TO BE AN INFECTIVE SINUS RETICULOSIS PRODUCED BY THE RICKETTSIAE. THE REACTIONS SEEN IN THE BOWEL WERE LOCAL AND, AS FAR AS ONE COULD JUDGE, INCONSTANT.

IN MY CASES, THE MORBID ANATOMICAL APPEARANCES OF SIXTEEN CASES AND MORBID HISTOLOGICAL APPEARANCES OF THREE CASES DID NOT SUGGEST THAT MYOCARDITIS AND INTERSTITIAL MYOCARDITIS WAS A COMMON CAUSE OF DEATH. SETTLE, PINKERTON AND CORBETT (283) CONSIDERED MYOCARDITIS TO BE A CAUSE OF DEATH IN FIFTY PER CENT OF FIFTY-FIVE CASES OF SCRUB TYPHUS. ALLEN AND SPITZ (2) HAVE SUGGESTED THAT ADRENAL CORTICAL DAMAGE IS RESPONSIBLE FOR SOME OF THE SYMPTOMS SEEN IN SCRUB TYPHUS. IN ONE SECTION EXAMINED I DID NOT FIND ANY OF THE CHANGES WHICH THEY DESCRIBED. THE CENTRAL NERVOUS SYSTEM LESIONS IN SCRUB TYPHUS ARE SCANTY AND DIFFICULT TO DEMONSTRATE AS LEWTHWAITE (147) HAS POINTED OUT.

TOXAEMIA HAS BEEN ASSUMED TO BE A CAUSE OF DEATH IN TYPHUS. FURTHER INFORMATION
ABOUT THE TOXIN OF RICKETTSIA ORIENTALIS IS REQUIRED. THE
CAUSE OF DEATH WOULD APPEAR IN MANY CASES TO BE ANOXAEMIA
FROM PNEUMONITIS, POSSIBLY DISTURBANCE OF EXCRETION FROM
NEPHRITIS, WITH POSSIBLY IN SOME CASES, CARDITIS, AND
PROBABLY "TOXAEMIA". IT SEEMS OBVIOUS THAT FURTHER
INVESTIGATIONS ARE REQUIRED BEFORE AN EXACT UNDERSTANDING
OF THE CAUSE OF DEATH CAN BE POSTULATED.

FURTHER IT IS OBVIOUS THAT SPECULATIONS AS TO THE
CAUSE OF DEATH ARE OF CONSIDERABLE CLINICAL IMPORTANCE,
IF RATIONAL THERAPEUTIC MEASURES ARE TO BE ADOPTED.

NO PATHOLOGICAL INVESTIGATIONS HAVE BEEN CARRIED
OUT ON THE OTHER TYPHUS LIKE FEVERS IN INDIA. THE ONLY
REPORTED AUTOPSY IS BY SACHS (270) OF A CASE WHICH MIGHT
BE CLASSIFIED AS OF THE INDETERMINATE SEROLOGICAL GROUP,
THOUGH SCRUB TYPHUS IS NOT IMPOSSIBLE.

AN IMMENSE FIELD FOR ACCURATE INVESTIGATION IS OPEN
IN INDIA AND BURMA. IT IS UNLIKELY HOWEVER THAT FURTHER
ADVANCES WILL BE MADE WITHOUT CONSIDERABLE RESEARCH, IN
WHICH HOWEVER THE CLINICIAN HAS A CONSIDERABLE PART
TO PLAY.
SUMMARY

(1) A SHORT HISTORICAL SURVEY OF THE DEVELOPMENT OF THE KNOWLEDGE OF TYPHUS AND TYPHUS-LIKE FEVERS AND A DESCRIPTION OF THE MAIN GROUPS IS PRESENTED.

(2) THE WEIL-FELIX TEST AND ITS NATURE AND APPLICATIONS IS DISCUSSED.

(3) THE LITERATURE OF TYPHUS IN INDIA AND BURMA IS SURVEYED.

(4) A DESCRIPTION IS GIVEN OF ONE HUNDRED AND FOURTEEN CASES SEEN PERSONALLY IN CALCUTTA, AND SOME PATHOLOGICAL FINDINGS IN SIXTEEN CASES.

(5) THE PATHOLOGICAL FINDINGS ARE BRIEFLY DISCUSSED.

(6) THE RECENT FINDINGS DURING THE SECOND WORLD WAR ARE CORRELATED AS FAR AS IS POSSIBLE WITH THE PRE-WAR INDIAN LITERATURE, AND THE PERSONAL FINDINGS IN CALCUTTA ARE COMPARED WITH FINDINGS ELSEWHERE, AND THE OBVIOUS GAPS IN PRESENT KNOWLEDGE ARE INDICATED.
REFERENCES.

(1) AGRESS, C.M. AND EVANS, E.R.
CLINICAL SURVEY OF 86 CASES OF SCRUB TYPHUS.
BULL. U.S. ARMY MED. DEPT. NO.2 p.553-569, FEB. 5' 1946.
ABSTRACTED IN TROP. DIS. BULL. VOL.43 NO.6 p.549 JUNE 1949.

(2) ALLEN, A.G. AND SPITZ, SOPHIE.
A COMPARATIVE STUDY OF THE PATHOLOGY OF SCRUB TYPHUS
(TSUTSUGAMUSHI DISEASE) AND OTHER RICKETTSIAL DISEASES.

(3) ANDERSON, J.F. AND GOLDBERGER, J.
THE EXPERIMENTAL PROOF OF THE IDENTITY OF BRILL'S DISEASE
AND TYPHUS FEVER.
NEW YORK MED. JL.95.1 MAY II p.976 1912.

(4) ANDREW, R.
FLUID BALANCE IN SCRUB TYPHUS.
MED. JL. AUSTRALIA. VOL.2 NO.24 p.432-434 DEC.5' 1945

(5) AMICSTEIN, I.
STUDIES FROM THE INSTITUTE FOR MEDICAL RESEARCH FEDERATED
MALAY STATES. NO.22. RESEARCHES ON TROPICAL TYPHUS. A STUDY
OF THE BACTERIOLOGY, SEROLOGY AND EPIDEMIOLOGY OF THAT
DISEASE.

(6) ANDERSON, S.V.W. REFERRED TO BY WIGAW AND SUNDER RAO
NOTES FROM SEVERAL CASES OF PSEUDO- OR PARA-TYPHOID.
KENYA MED. JL. VOL.2 p.42, 1925.
(7) ANIGSTEIN, L. AND BADGER, M. N.
ANTI-RICKETTIESIAL ACTIVITY OF PARA-AMINO-BENZOIC ACID (PABA):
EFFECT OF PABA ON EXPERIMENTAL SPOTTED FEVER.
TEXAS REPORTS ON BIOLOGY AND MEDICINE. VOL. 4 NO. 2 p. 280-
278, 1946. ABSTRACTED IN TROP. DIS. BULL. VOL. 43 NO. II p. 1036
NOV. 1946.

(8) ANNUAL REPORTS, DIRECTOR OF PUBLIC HEALTH, INDIA, 1943.
QUOTED BY CHALCROFT, W. S. AND BAKER, A. B. (47)

(9) ARKWRIGHT, J. A. AND BACOT, A. W.
TYPHUS FEVER.

(10) BADGER, L. F.
ROCKY MOUNTAIN SPOTTED FEVER AND RIOGRANEXUE FEVER: STUDY OF
THEIR IMMUNOLOGICAL RELATIONSHIP.

(11) BADGER, L. F., DYER, R. E. AND RUMREICH, A.
AN INFECTION OF THE ROCKY MOUNTAIN SPOTTED FEVER TYPE.
IDENTIFICATION IN THE EASTERN PARTS OF THE UNITED STATES.

(12) BALFOUR, A.
SO CALLED PSEUDO TYPHUS.
REFERED TO BY PIJPER, A. AND DAU, H. (247)

(13) BALMR SINGH.
A REPORT ON THIRTY CASES OF TYPHUS FEVER (LOUSE BORNE).
IND. MED. GAZ. VOL. 79, P. 146, APR. 1944.
(15) BANERJI, R.N.
A CASE OF TICK TYPHUS AT ALLAHABAD.
IND. MED. Gaz. Vol. 73; p. 297; May, 1937.

(16) BARDAN, P.N.
TYPHUS IN THE UNITED PROVINCES OF INDIA; BEING A CONTRIBUTION TO THE STUDY OF TYPHUS FEVER BASED ON A PAPER READ AT A CONFERENCE OF PATHOLOGISTS IN THE MIDDLE EAST AT CAIRO, ON 5 MARCH.
IND. MED. Gaz. Vol. 79; p. 150; Apr. 1944.

(17) BASU, U.P.
QUOTING DR. SATYA SARAN MTRTA OF HOWRAH. REMARKS IN DISCUSSION OF A PSEUDO TYPHUS EPIDEMIC IN SOUTHERN QUEENSLAND AND ITS EPIDEMIOLOGICAL BEARING ON CASES IN INDIA.
TRANS. OF 7TH CONG. FAR EAST ASS. TROP. MED. THACKER'S PRESS AND DIRECTORIES LTD. CALCUTTA. pp. 517-540; DEC. 1927.

(18) BASU, U.P.
FIFTEEN CASES OF EXANTHEMATIC TYPHUS IN CALCUTTA.
IND. MED. Gaz. Vol. 59; p. 396; Aug. 1924.

(19) BENGSTON, I.A. AND TOPPING, N.N.
THE SPECIFICITY OF THE COMPLEMENT FIXATION TEST IN ENDemic TYPHUS USING A RICKETTSIAL ANTIGEN.

(20) BEVERIDGE, A.J. AND UNDERHILL, E.
NOTES ON A CASE OF JAPANESE FEVER FEVER AND A CASE OF TROPICAL TYPHUS.

(21) BICKAM, J.
THREE CASES OF TROPICAL TYPHUS IN BANGALORE.
J. R. A. M. C. Vol. 59; p. 96; 1932.
(22) BLANCO, G. AND BALLHAZARD, M.
LONGEVITÉ DU VIRUS DE TYPHUS MULTIN DANS LES DEPOSITIONS DE PU
PO CES INFECTIONS.
BULL. SOC. PATH. EXOT. VOL. 33 NO. X PP. 95-32, JAN. 30° 1940.
(23) BLANCO, G. AND BALLHAZARD, M.
INTRODUCTION GÉNÉRALE ENTRE LA FIÈVRE BOUTONNEUSE ET LA FIÈVRE
POURPRE DES MONTAGNES NOGREUSE. VACCINATION DU COYAYE CONTR
CES DEUX MALADIES PAR UN VIRUS VOUTANT BILIE DE FIÈVRE
BOUTONNEUSE.
CR. REND. SC. VOL. 265,14, PP. 578-580, OCT. 4° 1932.
(24) BLAKE, F.C., MAXEY, K.F., SADUSK, J.F., KOHLS, G.M. AND
BELL, J.E.
STUDIES ON TSUTSGAMUSHI DISEASE (SCRUB TYPHUS) (MITE-BORNE
TYPHUS) IN NEW GUINEA AND ADJACENT ISLANDS: EPIDEMIOLOGY,
CLINICAL OBSERVATIONS, AND ETIOLOGY IN THE DOBADURA AREA.
AMER. J. HYG. VOL. 41, P. 243, MAY 1945.
(25) BLEWITT, B.
REVIEW OF FEVERS OF THE TYPHUS GROUP (VECTOR UNKNOWN)
OCcurring AT AHMEDNAGAR DURING 1933.
(26) BLEWITT, B.
FEVERS OF THE TYPHUS GROUP IN THE BHIM TAL AREA, KULAKU
HILLS U.P., INDIA. BEING A REPORT OF AN INVESTIGATION CARRIED
OUT INTO THE ALLEGED INCIDENCE AND NATURE OF TYPHUS GROUP
FEVERS IN THE BHIM TAL AREA, KULAKU HILLS, JULY 1936.
(27) BOYES-SMITH, D.
QUOTED BY CHEVERS (48).
MEDICAL TIMES AND GAZETTE P. 121 AUGUST 1879.
(28) BOYD, J. S. K.
FEVERS OF THE TYPHUS GROUP IN INDIA. AN ANALYSIS OF 110 CASES REPORTED IN 1934.

(29) BRADLEY, F. H. AND SMITH, F.
A CURIOUS CASE OF FEVER IN CALCUTTA.
JL. R.A.M.C. VOL. 19, P. 219, 1912.

(30) BRIDGES, R. F.
TROPICAL TYPHUS AND THE WEILL-FELIX REACTION.
JL. R.A.M.C. VOL. 62 P. 102, 1934.

(31) BRIDGES, R. F.
The Preparation of Suspensions For the Weill-Felix Test.
JL. R.A.M.C. VOL. 64, P. 153, 1935.

(32) BRIDGES, R. F.
Note on the Preparation of Suspensions For the Weill-Felix Test.
TRANS. R. SOC. TR. MED. & HYG. VOL. 37, NO. 5 P. 343, MARCH 1944.

(33) BRINGAL, G. D. AND BENSTON, I. A.
A STUDY OF THE COMPLEMENT FIXATION AND WEILL-FELIX REACTIONS IN WILD RATS AS RELATED TO THE ISOLATION OF THE VIRUS OF ENDEMIC TYPHUS.

(34) BRILL, N. E.
A STUDY OF SEVENTEEN CASES OF A DISEASE CLINICALLY RESEMBLING TYPHOID FEVER BUT WITHOUT THE WIDAL REACTION.
NEW YORK MEDICAL JOURNAL, VOL. 67, PP. 48 & 77: 1906.
(35) BRILL, N.E.
AN ACUTE INFECTIONOUS DISEASE OF UNKNOWN ORIGIN. A CLINICAL
STUDY BASED ON 281 CASES.

(36) BRITISH ENCYCLOPEDIA OF MEDICAL PRACTICE.
EDITED BY SIR HUMPHRY ROLLESTON. VOL. 12, pp. 335 - 350.
BUTTERWORTH'S MEDICAL PUBLICATIONS.

(37) BROWNING, J.S.; RAFHAEL, M.; KLEIN, E.F. AND GOLDBEZ, A.
SCRUB TYPHUS.

(38) BUCHWALD, HILDEGARD.
UNTERSUCHUNGEN UBER NORMALAGGLUTININE GEGEN PROTEUS X STÄRME.
ZTCHR. F. IMMUNITATSFR. U EXPERM. THERAP. TOME 99, NO. 6,
pp. 409-418, MAY 12, 1941. SUMMARY IN TROP. DIS. BULL.
VOL. 39, p. 137 1942.

(39) BUCKLAND, F.R.; DUDGEON, A.; EDWARD, D.C.H.; HENDERSON-BEGG,
A.; HAGHALLUM, F.O.; NIVEN, J.S.; F.; ROWLANDS, I.W.; AND VAN DEN
ENDE, M., WITH H.E. BARGEMANN, E.E. CURTIS, AND M.A. SHEPHERD.
SCRUB TYPHUS VACCINE: LARGE SCALE PRODUCTION.

(40) BURNET AND OLMER.
LA MALADIE DE MARSEILLE.
ARCHIVES DE L'INSTITUT PASTEUR DE TUNIS, Tome 16, NO. 4,
DEC. 1927.

(41) BUSH, F. KEITH.
TYPHUS FEVER IN SIMLA HILLS.
(42) CAFFARENA, W.M.
PATHOLOGISCH ANATOMISCHE BETILIGUNG DER NIREN BEIM FLECKFIEBER. ABSTR. ZENTRALEBL. F. ALLG. PATH. ANAT. VOL. 67, p. 203, 1937. QUOTED BY ALLEN, A.C. AND SPITZ, S. (2)

(43) CALDER, R.M.
BRUCELLA, PASTURELLA TULARENSIS AND PROTEUS AGGLUTININS IN CHRONIC BRUCELLOSIS.
JL. BACT. VOL. 41, NO. 5, PP. 593-604, MAY 1941.

(44) CASTANEDA, M.R.
STUDIES IN THE MECHANISM OF IMMUNITY IN TYPHUS FEVER.

(45) CASTANEDA, M.R. AND ZIA, S.
THE ANTIGENIC RELATIONSHIP BETWEEN PROTEUS X19 AND TYPHUS RICKETTSIAE. A STUDY OF THE WEIL-FELIX REACTION.
JL. EXPT. MED. VOL. 58, P. 55, 1933.

(46) CHELIN, W. (QUOTED BY ALLEN, A.C. AND SPITZ, S. (2))
DIE PATHOLOGISCHE ANATOMIE DES FLECKFIEBERS.

(47) CHALGREN, W.S. AND BAKER, A.W.
GENERAL REVIEWS.
TROPICAL DISEASES. INVOLVEMENT OF THE NERVOUS SYSTEM.
FROM THE DEPT. OF NEUROPSYCHIATRY, THE MEDICAL SCHOOL, UNIVERSITY OF MINNETOSA.
ARCHIVES OF PATHOLOGY, P. 66, JAN. 1946.

(48) CHEVERS, DR.
MEDICAL TIMES AND GAZETTE, P. 181, AUG. 1879.
(49) CHRISTIAN, C.R.
A CASE OF TYPHUS DUE TO TICK BITE
(50) CHRISTIAN, C.R.
THE WEIL-FELIX REACTION IN FEVERS OF UNCERTAIN ORIGIN.
(51) CONOR, A. AND BRUCH, A.
UNE FIEVRE ERUPTIVE OBSERVEE EN TUNISIE.
(52) COVEL, G.
STUDIES ON TYPHUS IN THE SIMLA HILLS; INTRODUCTION.
IND. JL. MED. RESEARCH. VOL. 23, p.701-706. JAN. 1936.
(53) COVEL, G.
STUDIES ON TYPHUS IN THE SIMLA HILLS; WEIL-FELIX
REACTION IN WILD RATS.
(54) COVEL, G.
STUDIES OF TYPHUS IN THE SIMLA HILLS; STRAIN OF TYPHUS
RECOVERED FROM WILD RATS.
IND. JL. MED. RESEARCH. VOL. 23, pp.713-720, JAN. 1936.
(55) COVEL, G. AND METHA, D.R.
STUDIES OF TYPHUS IN THE SIMLA HILLS; ROLE OF RAT FLEA
IN TRANSMISSION OF TYPHUS.
(56) COVEL, G.
STUDIES OF TYPHUS IN THE SIMLA HILLS; ATTEMPTS TO ESTABLISH
STRAINS OF TYPHUS FROM HUMAN SOURCES.
IND. JL. MED. RESEARCH. VOL. 24, pp.139-149, JULY 1936
(57) COVEL'L.C. AND METHA.D.R.
STUDIES ON TYPHUS IN THE SIMLA HILLS; ROLE OF HUMAN BODY LOUSE IN TRANSMISSION OF TYPHUS.
IND. Jl. MED. RESEARCH, VOL. 24, PP. 388-397, OCT. 1936.

(58) COWDRY,E.V.
THE DISTRIBUTION OF RICKETTSIA IN THE TISSUES OF INSECTS AND ARACHNIDS.
JL. EXPT. MED. VOL. 37, Ff. 431-456, 1923.

(59) COWDRY,E.V.
RICKETTSIAE AND THEIR RELATIONSHIP TO DISEASE.
ARCH. PATH. LAB. MED. 2, 59-90, 1926.

(60) CRAGG,F.W.
REMARKS ON THE TYPHUS FEVER OF KUMAON AND ON THE SUGGESTION THAT IT IS TRANSMITTED BY A TICK.
IND. MED. GAZ., VOL. 57, P. 291 AUS. 1922.

(61) CURRAN,E. J.
SOME OBSERVATIONS ON FEVER OF THE TYPHUS GROUP (VECTOR UNKNOWN).
JL. R. A. M.C., VOL. 56, P. 94, FEB. 1936.

(62) CUNNINGHAM,J. AND THEODORE,J.H.
TYPHUS FEVER IN THE AGENCY TRACTS, MADRAS PRESIDENCY.
IND. MED. GAZ., VOL. 59, P. 506, 1924.

(63) DAMKIN, G.J. AND BILLINGS,F.T. JR.
THE WEIL-FELIX REACTION IN PATIENTS WITH PROTEUS AND PSEUDOMONAS AERUGINOSA INFECTIONS.
JL. IMMUNOLOGY, VOL. 44, NO. 3, PP. 251-256, JULY 1942.
(64) Davies, J. W., and Johnson, W. B.

NOTES UPON THE OCCURRENCES OF A TWELVE-DAY FEVER OF DENQUE GROUP IN NIGERIA.


(65) Dawydowskie, J. W.

DIE PATHOLOGISCHE ANATOMIE UND PATHOLOGIE DES FLECKFIEBERS.


(66) D'Es. C.

A CASE OF SPOTTED FEVER (ROCKY MOUNTAIN FEVER; TICK FEVER OR TYPHUS FEVER).


(67) Delmore, F.

LES FIEVRES TYPHO-ÉXANTHEMAIQUES EN INDOCHINE MERIDIONALE.


(68) Derrick, E. H.

Q FEVER; A NEW FEVER ENTITY: CLINICAL FEATURES, DIAGNOSIS AND LABORATORY INVESTIGATION.


(69) Deshmukh, M. D.

CLINICAL PICTURE OF SCRAUB TYPHUS IN EAST AFRICAN TROOPS ON THE BURMA FRONT.


(70) Dick, J. C.

NOTES ON THE WEIL-FELIX REACTION IN TYPHUS FEVER AND OTHER DISEASES.

(71) DOWDEN, R.
A SUSPECTED CASE OF KEDANI RIVER FEVER IN THE FEDERATED MALAY STATES.

(72) DURAND, P. AND SPARROW, H.
INNOCULATION PULMONAIRE DES VIRUS TYPIQUES ET BOUTONNEUX.
COMPT. REND. ACAD. SCI., 210, 420: 1940.

(73) DYER, R.E.,CEDER,E.T.,RHIREICH,A. AND BADGER,L.F.
EXPERIMENTAL TRANSMISSION OF ENDEMIC TYPHUS FEVER OF UNITED STATES BY RAT FLEA (XENOPHILLA CHEOPS).

(74) DYER, R.E.,CEDER,E.T., WORKMAN, W.G.,RHIREICH,A. AND BADGER,L.F.
TYPHUS FEVER: TRANSMISSION OF ENDEMIC TYPHUS BY RUBBING EITHER CRUSHED OR INFECTED FLEAS OR INFECTED FLEA FANGS INTO WOUNDS.

(75) EDITORIAL: TROPICAL TYPHUS.
IND. MED. GAZ., VOL. 71, PP. 213 - 215, APRIL 1936.

(76) EDITORIAL: CYSTICERCOSIS IN MAN.
IND. MED. GAZ., VOL. 71, PP. 341, JUNE 1936.

(77) ELSDON-DREW, R.
RELAPSING FEVER AND B. PROTEUS X KINGSBURY.
NATURE, VOL. 152, P. 565: 1943.

(78) EVANS, C. LOVATT,
RECENT ADVANCES IN PHYSIOLOGY, 6th. ED., PP. 30 - 31, 1939.
(79) EWING, E. R.
THE TROMBICULID MITES (CHRIGER MITES) AND THEIR RELATION TO DISEASE.

(80) FAIRLEY, N. HAMILTON,
ARTICLE ON MITE TYPHUS IN A TEXTBOOK OF THE PRACTICE OF MEDICINE EDITED BY F.W. PRICE, 7th ED., SECOND IMP. NOV. 1947.

(81) FAIRWEATHER, QUOTED BY HUSBAND, J. AND MCWATTERS, R.C.

(82) FAIRQUAR, QUOTED BY HUSBAND, J. AND MCWATTERS, R.C. (I25)
NO REFERENCE GIVEN.

(83) FEJCIN, B.
SUR LA PERSEITANCE DU VIRUS DU TYPHUS EXANEMATIQUE DANS LES POUX.

(84) FELIX, A.
ON THE NON-SPECIFIC STIMULATION OF AGGLUTININS WITH SPECIAL REFERENCE TO THE ENTERIC FEVERS AND TYPHUS FEVER.
JL. HYG., VOL. 26, pp. 416-446, FEB. 1929.

(85) FELIX, A.
SPECIFIC AND NON-SPECIFIC SERUM REACTION IN TYPHUS FEVER.

(86) FELIX, A.
THE SEROLOGY OF THE TYPHUS GROUP OF DISEASES.

(87) FELIX, A.
TECHNIQUE AND INTERPRETATION OF THE WEIL-FELIX IN TYPHUS.
TRANS. ROY. SOC. TR. MED. & HYG., VOL. 37, NO. 5, P. 321, MAR. 1944.
(88) FELIX, A.

DISCUSSION ON PAPER - "OBSERVATIONS ON TSUTSUGAMUSHI DISEASE (SCRUB TYPHUS) IN ASSAM AND BURMA" BY MACKIE, TT. (169)

TRANS. ROY. SOC. TR. MED. & HYG., VOL. 40, NO. 1, P.55,

(89) FELIX, A.

PERSONAL COMMUNICATION TO MEGAW, J. D. W., ON SOME ANOMALOUS WEIL-FELIX REACTIONS.


(90) FELIX, A. AND OLITSKI, L.

THE USE OF PRESERVED BACTERIAL SUSPENSIONS FOR THE AGGLUTINATION TEST WITH SPECIAL REFERENCE TO THE ENTERIC FEVERS AND TYPHUS FEVER.

JL. HYG. VOL. 28, PP. 55 - 56, 1928.

(91) FELIX, A. AND RHODES, M.

SEROLOGICAL VARIETIES OF TYPHUS FEVER.

JL. HYG. VOL. 31, 225, 1931.

(92) FERNANDO, G. MENTIONED BY NICHOLS, L. (219).

(93) FINDLAY, G. M.

LABORATORY INVESTIGATIONS ON TYPHUS.

PR. ROY. SOC. MED. VOL. 35, PT. 1, P. 157, 1941 - 1942.

(94) FINDLAY, G. M.

PNEUMONITIS IN MICE, INFECTED INTRANASALLY WITH Q FEVER.

TRANS. ROY. SOC. TROP. MED. & HYG. VOL. 35, NO. 4, P. 213, JAN. 1942.

(95) FLETCHER, W. AND LESSLAR, J. E.

TROPICAL TYPHUS IN THE FEDERATED MALAY STATES.

BULL. INST. MED. RES., F. M. S., NO. 2, 1925.

JOHN BALE, SONS AND DANIELSSON LTD., LONDON, 1925.
(96) FOSHAY, L.
THE LABORATORY DIAGNOSIS OF UNDULANT FEVER.
AMER. JL CLIN. PATH. VOL. 10: p. 176, 1940.

(97) FRANKEL, E.
UBER FLECKFIBBER U ROSEOLA.
MUNCH. MED. WOCH., VOL. 51, NO. 2, p. 57, 1914.

(98) FRASCATORIOUS, HIERONIYMI.
DE CONTAGIONE ET CONTAGIOSIS MORBIS. VENICE.
TRANSLATED BY W.C. WRIGHT, 1930, QUOTED BY MINSCH, A. (126).

(99) FREIDMAN, G.A.
BRILL'S SYMPTOM COMPLEX: TYPHUS FEVER; MANCHURIAN TYPHUS.
ARCH. INT. MED. VOL. 8: p. 427, 1911.

(100) FULTON, F. AND JOYNER, L.
CULTIVATION OF RICKETTSIA TSUTSUGAMUSHI IN LUNGS OF RODENTS.
PREPARATION OF A SCRUB TYPHUS VACCINE.
LANCET, DEC. 8th pp. 729 - 734, 1945.

(101) GATER, B. A. R.
ENTOMOLOGICAL INVESTIGATION IN RELATION TO TROPICAL TYPHUS IN MALAYA.
TRANS. OF 8th CONG. FAR EASTERN ASS. OF TROP. MED. SIAM.
2: 132, 1930.

(102) GEAR, J. AND DE MEILLON.
MURINE TYPHUS IN NATAL; LABORATORY INVESTIGATIONS; (REPORT OF ISOLATION OF VIRUS OF MURINE TYPHUS FROM RATS, RAT FLEAS AND FROM A HUMAN CASE).
SOUTH AF. MED. JL., VOL. 13, DEC. 23th, pp. 804 - 806, 1939.

(103) GERHARD, W.W.
ON THE TYPHUS FEVER, WHICH OCCURRED AT PHILADELPHIA IN THE SPRING AND SUMMER OF 1936.
Continued...


(Grose, C.)

Typhus-like fever (col. Megaw's tick typhus).


(Goyal, R.K.)

The presence of an epizootic of rickettsial infection in wild rats of Calcutta.


(Greenfield, J.G. and Carnichael, E.A.)

The cerebro-spinal fluid in clinical diagnosis.

Macmillan and Co Ltd., p. 129, 1925.

(Griffiths, J.T. Jr.)

A scrub typhus (Tsutsugamushi) outbreak in Dutch New Guinea.


(Guthier, C.E.H.)

Endemic typhus in New Guinea; its occurrence and probable vector.


(Gurbuksh Singh)

Report on an epidemic of scrub typhus (K form) treated at a general hospital in Burma.


(Hamilton, H.L.)

Effect of p-aminobenzoic acid on growth of rickettsiae and elementary bodies, with observations on mode of action.

(III) HASS, C.M. AND PINKERTON, R.
SPOTTED FEVER: EXPERIMENTAL STUDY OF FIEVRE BOUTONNEUSE.
JL. EXP. MED., VOL. 64, PP. 601 - 623: 1936.

(III2) HATORE, J.
ON THE ENDEMIC TSUTSUGAMUSHI DISEASE OF FORMOSA.

(III3) HAY, G.P.
SCRUB TYPHUS AT FORT X.

(III4) HAYASHI, N.
ETIOLOGY OF TSUTSUGAMUSHI DISEASE.

(III5) HEASLIE, W.G.
TSUTSUGAMUSHI DISEASE IN NORTH QUEENSLAND AUSTRALIA.
MED. JL. AUS., I: P. 380: 1941.

(III6) HEILIG, R.
TYPHUS IN RAJRUTANA.
IND. MED. GAZ., VOL. 51, P. 399: OCT. 1946.

(III7) HEILIG, R. AND NAIDU, V.R.
ENDEMIC TYPHUS IN MYSORE.
IND. MED. GAZ., VOL. 76, P. 705: DEC. 1941.

(III8) HEILIG, R. AND NAIDU, V.R.
FURTHER EXPERIENCES ON ENDEMIC TYPHUS IN MYSORE.
IND. MED. GAZ., VOL. 77, P. 338: JUNE 1942.

(III9) HEILIG, R. AND NAIDU, V.R.
COMPLIMENT FIXATION TEST PERFORMED BY N.H. TOPPING, U.S.
PUBLIC HEALTH SERVICE, AND OTHER OBSERVATIONS IN MYSORE
TYPHUS.
(II9) CONTINUED

IND. MED. GAZ., VOL. 79, p. 154, APRIL 1944.

(120) HENDERSON, W. QUOTED BY HEGAW, J.D.W. (I93).

EDIN. MED. SUR. JL., 61: 201, 1844.

(121) HEPPEL, E. C.

AN OUTBREAK OF TYPHUS FEVER IN PESHAWAR.

IND. MED. GAZ., VOL. 43, p. 205, JUNE 1908.

(122) HIRSH, A.

HANDBOOK OF GEOGRAPHICAL AND HISTORICAL PATHOLOGY.

TRANSLATED BY CHARLES CREIGHTON.

NEW SYDENHAM SOC. LOND. VOL. I, p. 545, 1883.

(123) HONE, F.S.

A SERIES OF CASES CLOSELY RESEMBLING TYPHUS FEVER.

MED. JL. AUSTRALIA, 9th YEAR, NO. 1, JAN. 7th, pp. 1 - 13, 1922.

(124) HONE, F. S. AND BULL, L. B.

FURTHER SERIES OF CASES CLOSELY RESEMBLING TYPHUS FEVER.


(125) HUSBAND, J. AND MCWATERS, R. C.

TYPHUS FEVER IN NORTHERN INDIA.

IND. MED. GAZ., VOL. 43, p. 201, JUNE 1908.

(126) HUSSAIN, NOOR.

TYPHUS IN KASHMIR.

IND. MED. GAZ., VOL. 80, p. 133, MARCH 1945.

(127) JACKSON, D.S.

TYPHUS FEVER IN GILGIT.

IND. MED. GAZ., VOL. 80, p. 207, APRIL 1945.
(I28) JULLARD, J. AND HENAFF.
TRoubles du métabolisme hydrochlorique au cours des typhus épidémique et murin. CHLOROPHIE VASculaire et système RETICULO-ENDOTHELIAL CONSIDERATIONS GÉNÉRALES.
REV. SERV. DE SAN. MIL., IIO; pp. 197-266, FEB. 1939.

(I29) KAPILA, G.C. AND MAITRA, G.C.
A SEVERE CASE OF SCRUB TYPHUS.
IND. MED. GAZ., VOL. 73, p. 417, JULY 1937.

(I30) KAWAMURA, R. AND IMAGAMA, Y.
DIE FESTELLUNG DES ERREGERS BEI DER TSUTSUGAMUSHI KRANKHEIT.
CENTRALBL. F. BAKT.; PARAZIT.; U INFEKT. (I ABT. ORIG.) I82, 253: 1931.

(I31) KAWAMURA, R. AND YAMAGUCHI, M.
UBER DIE TSUTSUGAMUSHI KRANKHEIT IN FORMOSA, ZUGLEICH EINE VERGLEICHENDE STUDIE DERSELBEN MIT DER IN NORD JAPAN.

(I32) KAWAMURA, R. AND YAMAMIYA, G.
ON THE TSUTSUGAMUSHI DISEASE IN THE FESCADOES.

(I33) KAWAMURA, R., QUOTED BY ALLEN, A.C. AND SPITZ, S.(2).
STUDIES ON TSUTSUGAMUSHI DISEASE.
THE MED. BULL., UNIV. OF CINCINNATI, NO. 1, 2; VOL. 4, 1926.
SPOKESMAN PUBLISHING CO., CINCINNATI, OHIO 1926.

(I34) KEMP, H.A., WRIGHT, H.E. AND WAYNE, F.
QUOTED BY CALDER, R.M. (43).
SPECIFICITY OF WEIL-FELIX REACTION.
TEXAS STATE MED. JL., VOL. 29, pp. 278-281, 1933.
(135) KEATES, H.C.
NOTES ON A CASE OF TYPHUS-LIKE FEVER OCCURRING AT MURREE.
IND. MED. GAZ., VOL. 57, p. 101, MARCH 1922.

(136) KER, C.B.
KER’S INFECTIOUS DISEASES, REVISED BY CLAUDE HUNDE, p. 223.
3rd. ED. 1929, OXF. UNIV. PRESS.

(137) KITASHIMA, T. AND MIYAJIMA, M.
STUDIEN ÜBER DIE TSU'TSUGAMUSHI KRANKHEIT.
1918.

(138) KLEIN, H.S.
AN EPIDEMIC OF SCRUB TYPHUS.
JL. R.A.M.C., VOL. 85, NO. 4, PP. 187 - 190, OCT. 1945.

(139) KNEELELAND, Y. JR. AND SLEETANA, H.F.
CURRENT BRONCHO-PNEUMONIA OF UNUSAL CHARACTER AND UNDETERMINED
ETIOLOGY.
BULL. JOHN HOPKINS HOSP. VOL. 67, PP. 229 - 267, 1940.

(140) KOUWENAAR, W.
ONDERZOEKINGEN OVER SUMATRAANSCHE RICKETTSIOSEN.
XI. DE PATHOLOGISCHE ANATOMIE VAN DE MIJKOORTS BIJ DEN
MENSEN.
GENEESK TYDSCHR. V. NEDERL.- INDIE. VOL. 80, NO. 18,
PP. 1139 - 1140, APRIL 30TH: 1940. ABSTRACTED IN TROP.
DIS. BUL., VOL. 37, NO. 12, P. 846, DEC. 1940.

(141) KOUWENAAR, W. AND WOLF, J.W.
EXPERIMENTAL SUMATRAAN LITE FEVER IN GUINEA PIGS.
JL. INF. DIS., VOL. 55, P. 315, 1934.
KUNDU, M.L.
A CASE OF TYPHUS FEVER IN RANGOON.

LABARMADIE, V.
A TYPHUS-LIKE FEVER OCCURRING IN A DENGUE EPIDEMIC.

LANGAN, A.F. AND MATTHEWS, R.Y.
THE ESTABLISHMENT OF MOSSMAN, COASTAL AND OTHER PREVIOUSLY
UNCLASSIFIED FEVERS OF NORTH QUEENSLAND AS ENDEMIC TYPHUS.

LABARTHIE, R.
NOTE SUR LE MECANISME DE TRANSMISSION DU VIRUS DU TYPHUS
MURIN PAR VOIE DIGESTIVE.

LEWTHWAITE, R.
CLINICAL AND EPIDEMIOLOGICAL OBSERVATION ON TROPICAL TYPHUS
IN THE FEDERATED MALAY STATES.
BULL. INST. MED. RES., F.M.S., NO. 1, 1930.

LEWTHWAITE, R.
THE PATHOLOGY OF TROPICAL TYPHUS (RURAL TYPE) OF THE FEDERATED
MALAY STATES.

LEWTHWAITE, R. AND SAVOOR, S.R.
RECENT WORK ON THE TYPHUS-LIKE FEVERS OF MALAYA.
TRANS. ROY. SOC. TROP. MED. & HYG., VOL. 29, NO. 6, p. 561,
APRIL 1936.
(I49) LEWTHWAITE, R. AND SAVOOR, S.R.
THE TYPHUS GROUP OF DISEASES IN MALAYA. PT. I AND 2.
THE STUDY OF THE VIRUS OF RURAL TYPHUS IN LABORATORY ANIMALS.
BR. JL. EXP. PATH., VOL. 17, NO. 1, PP. 1 - 22, FEBRUARY 1936.

(I50) LEWTHWAITE, R. AND SAVOOR, S.R.
THE TYPHUS GROUP OF DISEASES IN MALAYA. PT. 3.
THE STUDY OF THE VIRUS OF URBAN TYPHUS IN LABORATORY ANIMALS.
BR. JL. EXP. PATH., VOL. 17, NO. 1, P. 23, FEBRUARY 1936.

(I51) LEWTHWAITE, R. AND SAVOOR, S.R.
THE TYPHUS GROUP OF DISEASES IN MALAYA. PT. 4.
THE ISOLATION OF TWO STRAINS OF TROPICAL TYPHUS FROM WILD RATS.
BR. JL. EXP. PATH., VOL. 17, NO. 3, PP. 208 - 214, JUNE 1936.

(I52) LEWTHWAITE, R., HODGKIN, E.P. AND SAVOOR, S.R.
THE TYPHUS GROUP OF DISEASES IN MALAYA. PT. 6.
THE SEARCH FOR CARRIERS.
BR. JL. EXP. PATH., VOL. 17, NO. 4, PP. 309 - 312, AUGUST 1936.

(I53) LEWTHWAITE, R. AND SAVOOR, S.R.
THE TYPHUS GROUP OF DISEASES IN MALAYA. PT. 7.
THE RELATION OF RURAL TYPHUS TO THE TSUTSUGAMUSHI DISEASE (WITH SPECIAL REFERENCE TO CROSS IMMUNITY TESTS).
BR. JL. EXPT. PATH., VOL. 17, NO. 6, PP. 448-450 (SEE P. 458)
DECEMBER 1936.

(I54) LEWTHWAITE, R. AND SAVOOR, S.R.
THE TYPHUS GROUP OF DISEASES IN MALAYA. PT. 8.
THE RELATION OF THE TSUTSUGAMUSHI DISEASE (INCLUDING RURAL TYPHUS) TO URBAN TYPHUS.
BR. JL. EXPT. PATH., VOL. 17, NO. 6, PP. 461 - 466, DECEMBER 1936.
(155) LEWTHWAITE, AND SAVOOR, S.R.

RICKETTSIAL DISEASES OF MALAYA. IDENTITY OF TSUTSUGAMUSHI AND RURAL TYPHUS.
LANCET, VOL. I, p. 255, FEBRUARY 10th, 1940.

(156) LEWTHWAITE, R. AND SAVOOR, S.R.

THE WEIL-FELIX REACTION IN EXPERIMENTAL RAT BITE FEVER.
NR. JL. EXPT. PATH., VOL. 22, p. 274, 1941.

(157) LEPINE, P., CAMINOPEETROS, J. AND PANGALOS, G.

PRÉSENCE DU VIRUS DU TYPHUS EXANTHEMATIQUE CHEZ LES PUCES DES RATS D'ATHÉNES ET FÍRÉE.
COMPT. REND. SOC. DE BIOL., VOL. 109, p. 710, 1932.

(158) LILIE, R.D.

PATHOLOGY OF ROCKY MOUNTAIN SPOTTED FEVER.
NAT. INST. OF HEALTH BULL., NO. 177, 1940.

PATHOLOGY OF THE EASTERN TYPE OF ROCKY MOUNTAIN SPOTTED FEVER.
U.S. PUB. HEALTH REP., VOL. 46, PART 2, PP. 2840-2859, 1931.

(159) LINDBERG, K.

UN CAS DE FÉVRE EXANTHEMATIQUE OBSERVEE DANS L'INDÉ BRITANNIQUE.

REVIEWED IN TR. DIS. BULL., VOL. 29, P. 19, 1932.

(160) LINDNER, S.J.L.

TWO CASES OF TROPICAL TYPHUS AND OTHER FEVERS.
JL. R. A. M. C., VOL. 69, P. 136, 1933.
(161) LITTLEJOHN, SIR HENRY

ADDRESS ON THE SANITATION OF EDINBURGH TO THE SECTION OF
STATE MEDICINE AT THE BRITISH MEDICAL ASSOCIATION MEETING,
EDINBURGH, 1898.

BR. MED. JL., 2: p. 401, AUGUST 13th, 1898.

(162) LIU, WEIC-T'UNG AND CHUNG, HUI-LAN.

MURINE TYPHUS VIRUS ISOLATED FROM A PATIENT IN PEIPING, CHINA.

PROG. SOC. EXPERIM. ETOL. AND MED., VOL. 40, NO. 3, PP. 350-353,
MARCH 1939. ABSTRACTED IN TR. DIS. BULL. VOL. 37, NO. 4,
P. 261, APRIL 1940.

(163) LOW, JOHN.

TEN CASES OF TYPHUS IN CIVILIANS IN CALCUTTA.

IND. MED. GAZ., VOL. 81, P. 171, APRIL-MAY 1946.

(164) LYELL - QUOTED BY HUSBAND, J. AND MCKATTERS, R.C.

IND. ANNS. MED. SC. 1852.

(165) LUSK, J.W.

ONE HUNDRED AND FOURTEEN CASES OF TYPHUS FEVER SEEN IN AN
INDIAN MILITARY HOSPITAL IN CALCUTTA.

IND. MED. GAZ., VOL. 60, NO. 9, PP. 437-445, SEPTEMBER 1945.

(166) MACARTHUR, W.F.

OLD TIME TYPHUS IN BRITAIN.

TRANS. ROY. SOC. TROP. MED. AND HYG., VOL. 20, NO. 8, P. 487,
1927.

(167) MACNAMARA, C.V.

EPIDEMIC OF TYPHUS IN SIMLA HILLS. (VECTOR UNKNOWN).


(168) MACKENZIE, L.H.L.

NOTES ON TWO CASES OF UNDIAGNOSED FEVER.

IND. MED. GAZ., VOL. 62, P. 699, 1927.
(169) Mackie, T. T., Davis, C. E., Fuller, H. S., Knapp, J. A.,
Steinacker, M. L., Stager, K. E., Traub, R., Jellison, W., Millsbaugh,
D. D., Austin, R. C., Bell, E. J., Kohls, C. M., Hsi, W., and
Girshman, J. A. W.

Observations on Tsutsugamushi Disease (Scrub Typhus) in
Assam and Burma: Preliminary Report.

Amer. J. Hyg., 43:195; 1946. and in

(170) Manifold, J. A.

Unpublished Figures quoted by Blewitt, B. (26).

(171) Manson-Bahr, P.


(172) Maister, M. and Miller, A.


(173) Maitra, C. C. and Sen Gupta, P. N.

A Note on Cases of Typhus Fever in Burma and Their
Distribution.


(174) Marriot, H. L.

Water and Salt Depletion.


(175) Martin, C. De C. and Anderson, L. A. P.

A Case of Tropical Typhus Serologically Related to
Scrub Typhus" of the F. M. S.

(176) MARCHANDER, - AND BIDEAU, -

NOTE SUR L'EPIDEMIOLOGIE DE LA FIEVRE EXANTHEMATIQUE OBSERVEE A BOARD DES NAVINES DE GUERRE.

REVUE D'HYGIENE ET DE MEDICINE PREVENTIVE. 52, NO. 5 pp353 - 364, MAI 1930.

(177) MAXCY, K.F.

CLINICAL OBSERVATIONS ON ENDEMIC TYPHUS (BRILL'S DISEASE) IN THE SOUTHERN UNITED STATES.

PUB. HEALTH REP., VOL. 41: PT. I, P. 1213, 1926.

(178) MAXCY, K.F.

AN EPIDEMIOLOGICAL STUDY OF ENDEMIC TYPHUS IN SOUTH EASTERN U.S.A.

U.S. PUB. HEALTH REP., VOL. 41, PT. 2, PP. 2967-2990, 1926.

(179) MCCANN, C.F.

OBSERVATIONS ON A TYPHUS EPIDEMIC.

LANCET, VOL. 265: 2, P. 535, OCTOBER 1943.

(180) MCGOVERN, V.

PATHOLOGICAL ASPECTS OF SCRUB TYPHUS IN NEW GUINEA.

MED. JL. AUSTRALIA, VOL. 2, NO. 5, PP. 146-149, AUGUST 4TH 1945.

(181) MCKENZIE, W.E. REPORTED BY MEGAW (187)

REPORT ON THE HEALTH OF BHM TAL AND SAT TAL.

GOVERNMENT PRESS, ALLAHABAD, 1913.

(182) MCLIMANS, W.F. AND GRANT, C.W.

QUOTED BY EDITORIAL BH. MED. JL. P. 495, APRIL 12TH 1947.

SCIENCE 105:161; 1947.

(183) MCHAUGHT, J.C.

PARATYPHOID FEVER IN SOUTH AFRICA.

JL. R. A. M. C., VOL. 16, PP. 505-507, 1911.
(184) MCROBERT, G. R.

SOME POINTS IN THE TREATMENT OF TYPHUS FEVER.

IND. MED. GAZ., VOL. 60, p. 318, JULY 1925.

(185) MCGATTERS, M.R.C.; REPORTED AS BANERJEE, R.N.

TWO CASES OF TYPHUS FEVER IN KUMAON.

IND. MED. GAZ., VOL. 62, p. 204, MAY 1927.

(186) MEGAW, J.D.W.

A CASE OF FEVER RESEMBLING BRILL'S DISEASE.

IND. MED. GAZ., VOL. 52, p. 15, JANUARY 1917.

(187) MEGAW, J.D.W.

A TYPHUS-LIKE FEVER IN INDIA—POSSIBLY TRANSMITTED
BY TICKS.

IND. MED. GAZ., VOL. 56, p. 361, OCTOBER 1921.

(188) MEGAW, J.D.W.

A NOTE ON THE TWELVE DAY FEVER OF NIGERIA.

IND. MED. GAZ., VOL. 56, p. 371, OCTOBER 1921.

(189) MEGAW, J.D.W.

THE TYPHUS GROUP OF FEVERS.

IND. MED. GAZ., VOL. 59, p. 66, FEBRUARY 1924.

(190) MEGAW, J.D.W.; SHETTLE, F.E.; AND ROY, D.N.

TYPHUS-LIKE FEVER—PROBABLY TICK TYPHUS IN CENTRAL INDIA.

IND. MED. GAZ., VOL. 60, p. 53, FEBRUARY 1925.

(191) MEGAW, J.D.W.

INDIAN TICK TYPHUS.

IND. MED. GAZ., VOL. 60, p. 56, FEBRUARY 1925.

(192) MEGAW, J.D.W., AND RAO, S. SUNDER.

TICK TYPHUS AND OTHER SPORADIC FEVERS OF THE TYPHUS GROUP.

IND. MED. GAZ., VOL. 63, p. 306, JUNE 1928.
(193) Megaw, Sir John.

Typhus Fever in the Tropics.


(194) Megaw, Sir John W. D.


(195) Megaw, Sir John W. D.


(196) Megaw, Sir John W. D.


(197) Megaw, Sir John W. D.

Remarks in Abstract of Yeomans, A., Snyder, J. C., Murray, E. S., Ecke, R. S. and Zarafonetis, C. J. D. (349)


(198) Megaw, Sir John W. D.

Notes on Some Anomalous Weil-Felix Reactions.


(199) Metha, D. R.

Studies of Typhus in the Sierra Hills: Ectoparasites on Rats and Shrews with Special Reference to Their Possible Role in Transmission of Typhus.

(200) MENDELL, T.H.
SCRUB TYPHUS FEVER (TSUTSUGAMUSHI DISEASE) IN NEW GUINEA.
REPORT OF 75 CASES.
AMER. J. MED. SCI., VOL. 211, NO. 1, PP. 9-32, JANUARY 1946.
ABSTRACTED IN TR. DIS. BULL., VOL. 43, NO. 6, P. 548, JUNE 1946.

(201) MINCHIN, R.L.H.
VARIATIONS IN THE PLATELET COUNT IN TYPHUS ASSOCIATED
WITH HAEEMATOMA.
IND. MED. CIR., VOL. 73, P. 679, NOVEMBER 1938.

(202) MITHA, SATYA SARAN. QUOTED BY BASU, U.P. (17).
CALCUTTA MED. J., NOVEMBER 1937.

(203) MIYAJIMA, M. AND OKUMURA, T.
ON THE LIFE CYCLE OF 'AKAMUSHI', CARRIER OF NIIFON RIVER FEVER.
KITASATO ARCH. EXPER. MED., VOL. 1, NO. 1, PP. 1-14, APRIL 1917.

(204) MOOSER, H.
TABARDILLO, AN AMERICAN VARIETY OF TYPHUS.
JL. INF. DIS., VOL. 46, P. 126, 1930.

(205) MOOSER, H. AND DURMER, C.
EXPERIMENTAL TRANSMISSION OF ENDEMIC TYPHUS OF SOUTH EASTERN
ATLANTIC STATES BY THE BODY LOUSE.
JL. INF. DIS., VOL. 46, P. 170, 1930.

(206) MUKHERJEE, D.M.
A CASE OF TICK TYPHUS IN A TOWN.
IND. MED. CIR., VOL. 67, PP. 86-87, 1932.

(207) MUNK, F. QUOTED BY ALLEN, A. C., AND SPITZ, S. (2).
PATHOLOGIE UND KLINIK DER HILUSNERKRANKUNGEN.
URBAN UND SCHWARZENBERG, BERLIN UND WEIN, ED. 2, PP. 245-253, 1925.
MURCHISON, CHARLES.

A TREATISE ON THE CONTINUED FEVERS OF GREAT BRITAIN.
3rd Ed. Edited by W. Cayley. Longmans Green, 1864.

MURRAY, E.S., ZARAFONETIS, C.J.D., AND SNYDER, J.C.
FURTHER REPORT ON EFFECT OF PARAMINOBENZOIC ACID IN EXPERIMENTAL TSUTSUGAMUSHI DISEASE (SCRUB TYPHUS):
PROC. SOC. EXP. BIOL. AND MED., VOL. 60, NO. 1, PP. 80-84.
ABSTRACTED IN TROP. DIS. BULL., VOL. 43, NO. 5, P. 433, MAY 1946.

NAGAYO, M., TAKIYA, T., IMAMURA, A.; SATO, K., MIYAGAWA, Y., AND MITAMURA, T.
DEMONSTRATION OF THE VIRUS OF TSUTSUGAMUSHI DISEASE.
TRANS. JAP. PATH. SOC., VOL. 14, P. 193, 1924.

NAGAYO, M., MIYAGAWA, Y., MITAMURA, T., TAMIYA, T., AND TENJIN, S.
FIVE SPECIES OF TSUTSUGAMUSHI (THE CARRIER OF JAPANESE RIVER FEVER) AND THEIR RELATION TO THE TSUTSUGAMUSHI DISEASE.
AMER. J. HYG., VOL. 1, P. 569, 1921.

NAGAYO, M., MIYAGAWA, Y., MITAMURA, T., TAMIYA, T., SATO, K., HAZATO, H., AND IMAMURA, A.
ÜBER DEN NACHWEIS DES ERREIGNERS DER TSUTSUGAMUSHI-KRANKHEIT DER RICKETTSSIA ORIENTALIS.
JAPAN. J. EXP. MED., VOL. 9, NO. 2, PP. 67-150, MARCH 20TH 1931.

NAPIER, L.E.
THE PRINCIPLES AND PRACTICE OF TROPICAL MEDICINE.
THE MACMILLAN CO., NEW YORK 1946.

NAPIER, L.E.
EDITORIAL, IND. MED. GAZ., VOL. 71, P. 213, APRIL 1936.
(215) NAPIER, L.E. AND DAS GUPTA, G.R.
HAEMATOLOGICAL STUDIES. PART 3: NORMAL STANDARDS FOR A TEA-GARDEN
COOLIE POPULATION.
IND. JI. MED. RES., VOL. 23, P. 311, JULY 1ST 1935.

(216) NEIL, M. H.
EXPERIMENTAL TYPHUS FEVER IN GUINEA PIGS.
U.S. PUB. HEALTH REP., WASH., VOL. 32, NO. 28 P. 1105,
JULY 13TH 1917.

(217) NEUMBURGER, GEEVER, AND RUTLEDGE,
RHEUMATIC PNEUMONIA.
ARCH. PATH. VOL. 37, P. 15 1940.

(218) NICHOLS, L.
A CASE OF TSUTSUGAMUSHI (RURAL TYPHUS) IN CEYLON.
BR. MED. JI., 2: P. 490: 1940.

(219) NICHOLS, L.
REMARKS (P. 10) AT PROCEEDINGS OF A CONFERENCE ON AN OUTBREAK
OF SCRUB TYPHUS IN CEYLON. (258)

(220) NICOLLE, C.

(221) NICOLLE, C. NO REFERENCE, QUOTED BY STRONG, R.P. (304).

(222) NICOLLE C. AND ANDERSON, C.
RELAPSING FEVER TRANSMITTED BY BOTH TICKS AND LICE.
CONT. REND. ACAD. SC., VOL. 182, P. 1450, 1928.

(223) NICOLLE, C.; COMTE, C.; AND CONSEIL, E.
TRANSMISSION EXPERIMENTALE DU TYPHUS EXANTHEMATIQUE PAR LE FOU
DU CORPS.
COMPT. REND. DE L’ACAD DES SCI., VOL. 149, P. 486, SEPT. 6TH 1909.
(224) NICOLLE, C., CONSEIL, E. AND CONGO, A.
COMPT. REND. ACAD. D' SC., 152: p. 1632, 1911.

(225) NICOLLE, G. ET COMTE, C.
SUR LA PRESENCE FREQUENTE D'UN POUVOIR AGGLUTINANT VIS À VIS DU "M. MELITENSIS" DANS LE SANG DES MALADES ATEINTS DE TYPHUS EXANTHEMATIQUE, SA VALEUR DIAGNOSTIQUE.

(226) NICOLLE, CHARLES, GIROND, PAUL, ET SPARROW, HELENE.
PRESENCE OCCASIONNELLE DU VIRUS MURIN DANS LES URINES DE RATS INFECTES EXPERIMENTALEMENT PAR CE VIRUS.

(227) NICOLLE, CHARLES, GIROND, PAUL, ET SPARROW, HELENE.
PRESENCE EXCEPTIONNELLE DU VIRUS MURIN DANS LES URINES DE RATS INFECTES EXPERIMENTALEMENT PAR CE VIRUS.
ARCH. INST. PAST. DE TUNIS, VOL. 23, NO. 1, pp. 1-14, MAR. 1934.

(228) NICOLLE, C. ET LEBAILLY, C.
LES INFECTIONS EXPERIMENTALES INAPARENTES EXEMPLES TIRES DE L'ETUDE DU TYPHUS EXANTHEMATIQUE.
CONT. REND. ACAD. D' SC., 166: VOL. 1, p. 800: 1919.

(229) NORMAN, C. E. S. AND RAMACHANDRAN, C.S.
A CASE OF TICK TYPHUS.
IND. MED. GAZ., VOL. 60, p. 222, MAY 1925.

ARCH. INTERN. MED., VOL. 64, p. 105 1939.

(231) OGATA, N.
ANTIOLOGIE DER TSUTSUGAMUSHI-KRANKHEIT; RICKETTSIA TSUTSUGAMUSHI.
(232) Pai, M.N.
TWO CASES OF TICK FEVER FROM POONA.
IND. MED. GAZ., VOL 63, p. 704, DECEMBER 1928.

(233) Pai, M.N.
A CASE OF TICK TYPHUS IN POONA.
IND. MED. GAZ., VOL. 66, p. 200, APRIL 1931.

(234) Panayolalan, A.
LES RATS ROSEVOIRS DU TYPHUS EXANTHEMATIQUE A ALEXANDRE.

(235) Pandalai, N.G.
ENTERIC FEVER IN VIZAGAPATAM.
IND. MED. GAZ., VOL. 71, p. 511, SEPTEMBER 1936.

(236) Parker, M.T.
PERSONAL COMMUNICATION, WHILE OFFICER IN CHARGE DISTRICT
LABORATORY (FOR 14TH ARMY), CALCUTTA 1943.

(237) Parker, R.R. AND Davis, C.E.
PROTECTIVE VALUE OF CONVALESCENT SERA OF SAO PAULO EXANTHEMATIC
AGAINST VIRUS OF ROCKY MOUNTAIN SPOTTED FEVER.
JANUARY-JUNE 1933.

(238) Parker, R.R.
CERTAIN PHASES OF THE PROBLEM OF ROCKY MOUNTAIN SPOTTED FEVER.
ARCH. PATH., VOL. 15, p. 396, MARCH 1933.

(239) Parker, M.T. AND Savoor, S.R. QUOTED BY

(240) Parker, M.T. QUOTED BY
Zarafonitis, C.J.D. (352)
(241) PASRICA, C. L.; PANJA, G. AND LAL, S.

Immunological methods in the determination of infection in a random sample of hospital admissions. Part I (The frequency and concentration of 'H' and 'O' agglutinins of the typhoid-paratyphoid group in 260 individuals admitted into the Carmichael hospital for tropical diseases Calcutta).


(242) PASRICA, C. L., BANNERJEE, K. AND LAL, S.

Immunological methods in the determination of infections in a random sample of hospital admissions. Part 2 (The frequency and concentration of agglutinins for Proteus X strains in a series of hospital patients).


(243) PATEL, T. B.

*Typhus fever in Bombay.*


(244) PATEL, T. B.

*Further cases of typhus fever in Bombay City.*


(245) PHIPSON, E. S.

Observations on an outbreak of typhus in Simla with special reference to the Weil-Felix reaction.


(246) PHIPSON, E. S.

*Serological diagnosis of typhus by Weil-Felix reaction.*

(247) PIJPER, A. AND DAU, HELEN.
SOUTH AFRICAN TYPHUS.

(248) PINKERTON, H.
THE PATHOGENIC RICKETTSIAE.
BACT. REVIEWS. VOL.6, p. 37, MARCH 1942.

(249) PINKERTON, H.
MICROPHOTOGRAPHS OF LUNGS IN TOXOPLASMOSIS SUPPLIED FOR
ALLEN, A. C. AND SPITZ, S.'S ARTICLE (2).

(250) PINKERTON, H. AND HENDERSON, R.G.
A PREVIOUSLY UNRECOGNISED DISEASE ENTITY SIMULATING THE
TYPHUS-SPOTTED FEVER GROUP.

(251) PINKERTON, H. AND WEINMAN, D.
TOXOPLASMA INFECTION IN MAN.
ARCH. PATH., VOL. 30, pp. 374-392: 1940.

(252) PLAZY, MARCON ET CARBONI.
TYPHUS ENENDIQUE BENIN (MALADIE DE BRILL).

(253) PLOTZ, H.
COMPLIMENT FIXATION IN RICKETTSIAL DISEASES.
SCIENCE, VOL. 97, NO. 2505, p. 20, JUNE 1st 1943.

(254) PLOTZ, H. AND WERTMAN, K.
THE USE OF THE COMPLIMENT FIXATION TEST IN ROCKY MOUNTAIN
SPOTTED FEVER.
SCIENCE, VOL. 95, pp. 441-442, APRIL 24th., 1942.
MODIFICATIONS OF SEROLOGICAL RESPONSE TO INFECTION WITH MURINE
TYPHUS BY PREVIOUS IMMUNIZATION WITH EPIDEMIC TYPHUS VACCINE.
PROC. SOC. EXPER. BIOL. AND MED., VOL. 59, NO. 2, PP. 248-251,
JUNE 1945.

PRADHAN, K. C.
TYPHUS IN AKOLA (BERAR).
IND. MED. Gaz., Vol. 79, pp. 145, APRIL 1944.


PROCEEDINGS OF A CONFERENCE ON AN OUTBREAK OF SCRUB
TYPHUS IN GEYON, HELD AT H.Q. GEYON ARMY COMMAND 3RD FEB. 1944.

RAYNAL, J.
LE TYPHUS MURIN A SHANGHAI.
BULL. SOC. PATH. EXOT., VOL. 33, NO. 3, P. 168 MARCH 1940.

REIMAN, H. A., ULRICH, H. L. AND FISCHER, L. C.
DIFFERENTIAL DIAGNOSIS BETWEEN TYPHUS AND SPOTTED FEVER. REPORT
OF A CASE AND THE ISOLATION OF A NEW MILD TYPE OF SPOTTED FEVER
VIRUS.
JL. AMER. MED. ASS., VOL. 96, P. 1875: 1932.

REITLER, F., PLESH, S. AND MARBERG, K.
ENDEMIC TYPHUS IN PALESTINE.
TR. ROY. SOC. TROP. MED. & HYG., VOL. 33, NO. 2, P. 197, JULY 28th
1939.

RENARD, A. LE AND RAYNAUD, M.
SYNDROME NEPHROTOXIQUE SURAIGU DU TYPHUS EXANTHEMATIQUE.
DECAPSULATION DU REIN-QUERISON.
ARCH. MED. GEN. ET COLON., VOL. 8, NO. 3, PP. 77-83, 1939.
ABSTRACTED IN TR. DIS. BULL., VOL. 37, NO. 4, P. 258, APRIL 1940.
RICKETTS, H.T.

THE STUDY OF 'ROCKY MOUNTAIN SPOTTED FEVER' (TICK FEVER) BY MEANS OF ANIMAL INOCULATIONS.
JL. AMER. MED. ASS., VOL. 47, P. 33, 1906.

RICKETTS, H.T. AND WILDER, R.M.

JL. AMER. MED. ASS., VOL. 54, PP. 1304-1307, APRIL 16TH 1910.

ROBBINS, E.C., RAGAN, G.A., CAULD, R.I., WARE, F.B.,

RUSTIGIAN, R., SNYDER, M.J., SHADEL, J.W., FEINSTEIN, M., YESNER, R.,

MARKS, J.L., DINGLE, J.H., CHENEY, G., GEIB, W.A., TOPPING, N.H.,

SHEPARD, C.C., AND HUEBNER, R.J.

Q FEVER: A FOREWORD. INTRODUCTION TO A SERIES OF PAPERS DEALING WITH Q FEVER.

AMER. JL. HYG., NO. I PP. 1-5, NO. I 6-22, NO. I 23-50, NO. I 51-63,
NO. I 64-71, NO. I 72-87, NO. I 88-102, NO. I 103-109, NO. I 110-122,

ROCHA-LIMA, DA H.

BEOBACHTUNGEN BEI FLECKTYPHUS-LÄUSEN.

ARCH. SCHIFFS U TROP-HYG. 20:17; 1916.

ROSE, H.M.; DUANE, R.B. AND FISCHELL, E.E.

THE TREATMENT OF SPOTTED FEVER WITH PARA-AMINOBENZOIC ACID.
JL. AMER. MED. ASS., VOL. 129 NO. IV, PP. 1160-1161, DEC. 22nd 1945.

ROY, B.C.

TYPHUS FEVER, WITH SPECIAL REFERENCE TO ITS INCIDENCE IN INDIA.
JL. IND. MED. ASS., VOL. 15, NO. 5, PP. 135-146, FEBRUARY 1946.

ABSTRACTED IN TR. DIS. BULL., VOL. 43, NO. 6, P. 725, AUGUST 1946.
(269) SABIN, A.D.

TOKOPLASMIC ENCEPHALITIS IN CHILDREN.

JL. AMER. MED. ASS., VOL. 116, P. 801, 1941.

(270) SACHS, A.

NOTES ON SEVEN CASES OF THE INDIAN TYPHUS-LIKE FEVERS.

JL. R. A. M. C., VOL. 64, P. 163, 1935.

(271) SANGSTER, C.E. AND KAY, H.B.

SCRUB TYPHUS: CLINICAL ASPECTS.


(272) SARKAR, S.K.

A CASE OF TYPHUS FEVER.

IND. MED. GAZ., VOL. 74, P. 223, APRIL 1939.

(273) SAVOOR, S.R. REFERRED TO BY TATTERSAL, R.N. AND FARRY, T.R.

(311).

(274) SAVOOR, S.R. AND LEWTHWAITE, R.

THE WEIL-FELIX REACTION IN EXPERIMENTAL RAT BITE FEVER.

BR. JL. EXPERT. PATH., VOL. 22, P. 274, 1941.

(275) SAVOOR, S.R., CASTANEDA, M.R. AND ZINNSER, H.

NOTES ON THE WEIL-FELIX REACTION IN INDIVIDUALS NOT SUFFERING FROM TYPHUS.

PROC. SOC. EXPT. BIOI. & MED., VOL. 33, NO. 3, PP. 365 DECEMBER 1935.

(276) SAYERS, M.F.P.

REMARKS IN DISCUSSION ON PAPER "OBSERVATIONS ON TSUTSUGAMUSHI DISEASE (SCRUB TYPHUS) IN ASSAM AND BURMA: MACKIE, T.T. ET AL.

TRAN. ROY. SOC. TR. MED. GHYG., VOL. 40, NO. 1, PP. 50-52, AUGUST 1945.

(277) SCALES, C.

SOME OBSERVATIONS ON THE WEIL-FELIX REACTION.

(278) SCHÖFFER, W., QUOTED BY ALLEN, A.C. AND SPITZ, S. (3)
ZUR PATHOLOGIE DES FLECKFIEBERS.

(279) SCHUFTNER, W.

(280) SCHUFTNER, W. AND WACHMUTH, W.
ÜBER EINETYPHUS ARTIGE ERKANKUNG. (PSEUDO-TYPHUS VON DELI).

(281) SELLARDS, A.W.
THE CULTIVATION OF A RICKETYTA-LIKE MICRO-ORGANISM FROM
TSUTSUGAMUSHI DISEASE.

(282) SEN GUPTA, P.C.
A CASE OF TYPHUS FEVER COMPLICATING KALA AZAR.
IND. MED. GAZ., VOL.79, PP.602-603, DECEMBER 1944.

(283) SETTLE, E. B., PINKERTON, H. AND CORBETT, A.J.
A PATHOLOGIC STUDY OF TSUTSUGAMUSHI DISEASE (SCRUB TYPHUS) WITH
NOTES ON THE CLINOPATHOLOGIC CORRELATION.
JL. LAB. AND CLIN. MED., VOL.30, NO. 8, PP.639-661, AUGUST 1945.

(284) SEWARD, C.M., LATE OFFICER IN CHARGE, MEDICAL DIVISION
47 BRITISH GENERAL HOSPITAL, CALCUTTA; PERSONAL COMMUNICATION.

(285) SHARMA, L.R.
NON EPIDEMIC TYPHUS IN THE CIVIL POPULATION OF BANGALORE.
IND. MED. GAZ., VOL. 75, P.398, JULY 1940.

(286) SHORTT, H.E. AND D'SILVA, H.A.D.
THE DISTRIBUTION OF INDIAN TICK TYPHUS WITH NOTES ON
LABORATORY FINDINGS.
IND. MED. GAZ., VOL.71, P.13, JANUARY 1936.
(287) Silber, L. Quoted by Dauvin, G. J. & Billings, F. T. Jr. (63).

Über den Proteus "XV".


(288) Smadel, J. E., Rights, F. L. and Jackson, E. B.

Studies on scrub typhus. (1) Soluble antigen in tissues and body fluids of infected mice and rats.


(289) Smithson, O.


(290) Smith, F. K.

The use of para-aminobenzoic acid in endemic (murine) typhus;


(291) Smith, R. O. R. and Mehta, D. R.

Studies of typhus in the Simla Hills; attempts to isolate strains of Xk typhus from wild rats.


(292) Snyder, J. C. and Zarafovetis, C. J. D.

Effects of para-aminobenzoic acid in experimental tsutsugamushi disease (scrub typhus).


(293) Sommencchein, C.

Pseudo-Weil-Felix Reaktion bei Protexionen.


(294) Soni, R. L.

Typhus fever in Burma with record of three cases.

(295) SOUCHARD, MANEFTE, LIEU ET VIEILLE.
UN CAS DE FIEVRE PLUVIALE DU JAPON OBSERVE EN COCHINCHINE.
ETUDE CLINIQUE ET EXPERIMENTALE.
ARCHIVES DES INSTITUTS PASTEUR D'INDOCHINE. P.99, AVRIL 1932.
(296) SPARROW, H.
ESSAIS D'IMMUNIZATION AVEC LE VIRUS MURIN I DE TUNIS, INTRODUIT
PAR LA VOIE NASALE.
C. REND. ACAD. SC., VOL. 201, NO. 26, PP. 1441-1443, DEC. 23rd 1935.
(297) SPARROW, H., ET MARESCHAL, P.
REACTION DE L'ORGANISME HUMAIN A L'INOCULATION DES RICKETTSIA
DU VIRUS TYPhUS MURIN I DE TUNIS.
REV. D'IMMUNOLOGIE PARIS. VOL. 5, PP. 469-475, SEPT., 1939.
(298) SPENCER, R.R. AND PARKER, R.R.
ROCKY MOUNTAIN SPOTTED FEVER: INFECTIVITY OF FASTING AND
RECENTLY FED TICKS.
U.S. PUB. HEALTH REP., VOL. 36, NO. 8, REPRINT NO. 617
PP. 333-339, FEBRUARY 1923.
(299) SPENCER, R.R. AND PARKER, R.R.
ROCKY MOUNTAIN SPOTTED FEVER: VACCINATION OF MONKEYS AND MAN.
(300) STARYK, J.
VITALITE, TOXICITE ET POUVOIR D'IMMUNIZATION DE RICKETTSIA
PROWAZEKI CONSERVÉS HORS DE L'ORGANISME DU POU, EN MILIEU
LIQUIDE ET EN MILIEU SEC.
CONT. REND. SOC. BOL., VOL. 123, PP. 1221-1225; 1936.
(301) STEELE, J.M., MCLEISH, W.F., GRANT, C.W. AND TULLIS, J.L.
STUDIES OF TSUTSUGAMUSHI DISEASE (SCRUB TYPHUS), (5). THE EFFECT OF
METHYLTHIOIONINE CHLORIDE (METHYLTHYMINE BLUE) ON TSUTSUGAMUSHI DISEASE
IN MAN.
REPORT NO. 5, JL. NAV. MED. RES. INST. BETHESDA, MD. 1946.

(302) STILLE, A.
A TABLE OF COMPARISON BETWEEN TYPHUS AND TYPHOID FEVERS, 1838.
TRANSLATED BY W. PEPPER 1904. PHILADELPHIA.

(303) STOTT, H.
IMMUNOLOGICAL PROBLEMS OF THE TYPHUS GROUP AS RAISED BY A
SPORADIC CASE OF TYPHUS (VECTOR UNKNOWN) FROM HAMIRPUR IN THE
PLAINS OF INDIA WITH A NOTE ON THE HISTORY OF TICK TYPHUS
IN INDIA.
IND. MED. GAZ., VOL. 70, P. 325 APRIL 1935.

(304) STRONG, R.F.
EDITOR STITT'S DIAGNOSIS, PREVENTION AND TREATMENT OF TROPICAL
DISEASES. 7th ED., REPRINTED FEB. 1945, SEE p. 937.
THE BLAKISTON CO.

(305) STRUM, A.
DAS FLECKFIEBER UND SEINE BEDEUTUNG FÜR DIE KLINISCHE PATHOLOGIE
DES Stammls.
KLIN. WOCH., VOL. 21, NO. 41, PP. 899-904 OCTOBER 10TH 1942.

(306) SUNDER RAO, S.
The History of Tick Bites in Cases of Tick Typhus in India.
IND. MED. GAZ., VOL. 63, P. 314, JUNE 1928.
(307) TAMAKA, K.

ÜBER AETIOLOGIE UND PATHOGENESE DER KEDAMI-KRANKHEIT.
ZBL. BAKT., VOL. 26, p. 432; 1899.

(308) TATTERTSALL, R.N.

TSUTSUGAMUSHI FEVER ON THE INDIA-BURMA BORDER.
LANCET. pp. 392-394; SEPTEMBER 29th, 1945.

(309) TATTERTSALL, R.N. AND PARRY, T.E.

AN OUTBREAK OF TYPHUS FEVER (OXK) IN INDIA.
IND. MED. GAZ., VOL. 86, p. 433 SEPTEMBER 1945.

(310) THOMPSON, T.C.

FEVERS OF THE TYPHUS GROUP IN NORTHERN INDIA.
JL. R. A. M. C., VOL. 72 p. 267 APRIL 1939.

(311) TIERNEY, N.A.

EFFECTS OF PARA-AMINOBENZOIC ACID IN TSUTSUGAMUSHI DISEASE.
JL. AMER. MED. ASS., VOL. 131, No. 4 pp. 280-285, MAY 25th 1946.

(312) TOPELEY, W.W.C. AND WILSON, C.S.

THE PRINCIPLES OF BACTERIOLOGY AND IMMUNITY.
3rd. ED. EDWARD ARNOLD AND CO. LONDON.

(313) TOPPING, N.H.

ROCKY MOUNTAIN SPOTTED FEVER. A NOTE ON SOME ASPECTS OF ITS EPIDEMIOLOGY.
U.S. PUB. HEALTH REP., VOL. 56 NO. 34, pp. 1699-1703,
AUGUST 22nd, 1941.

(314) TOPPING, N.H.

TSUTSUGAMUSHI DISEASE (SCRUB TYPHUS). THE EFFECTS OF AN IMMUNE RABBIT SERUM IN EXPERIMENTALLY INFECTED MICE.
U.S. PUB. HEALTH REP., WASH., VOL. 60, NO. 41, pp. 1215-1220,
OCTOBER 12th 1945.
(315) TURNER, J.C., NISHEWITZ, S., JACKSON, E.B. AND HERNEY, R.
Relation of Cold Agglutinins to Atypical Pneumonia.

(316) VAUCEL, M. AND BRUNEAU, M.
ISOLEMENT, DES RATS DE HANOI, D'UNE SOUCHE DE PISTEUS OXK.

(317) VAN DEN ENDE, M., LOCKET, S., HARGREAVES, W.H.
NIVEN, J. AND LENINGHOFF, L.
ACCIDENTAL LABORATORY INFECTION WITH TSUTSUGAMUSHI RICKETTSIA.
Lancet. pp.4-7 July 6th 1946.

(318) VAN MEERENDONK, F.
ERFABRUCGEN UBER FLECKFEBREHANDLING MIT ATERBIN UND CALCIUM.

(319) VAN ROOYEN, C.E., AND BEARCHOFT, W.G.C.
TYPHUS RICKETTSIAL AGGLUTINATION TESTS IN THE MIDDLE FORCES
AND EGYPT.

(320) VIOLLE, H.
ETUDE EXPERIMENTALE DU TYPHUS EXANTHEMATIQUE SA TRANSMISSION
AVEC FIEVRE ET EXANTHEME CHEZ LE PORC.
Bull. Acad. Med. 3rd Ser., Vol. 118, No. 40, pp.809-812, Dec. 28th,
1937.

(321) VIOLLE, H.
CONTRIBUTION A L'ETUDE DU TYPHUS EXANTHEMATIQUE BURIN, DE LA
CONTAMINATION DU CHIEN PAR INGESTION DU VIRUS.

(322) VON PROWAZKCH.
BEITR. Z. KLIN. D. INFektIONSCHR., 4 p. 5 1915.
(323) WALCH, E.W. AND KEUKENSCHRIJVER, N.C.
EENIGE OPMERKINGEN Aangaande de epidemiologie van de
Pseudotyphus.
GENEESK. TIJDSCHR.V. NEDERL.-INDIE. VOL. 64, NO. 2, pp. 247-276, 1924.
ABSTRACTED IN TR. DIS. BULL.; VOL. 22, NO. 2, p. 122, FEB. 1925.

(324) WALKER, W.
ON AN EPIDEMIC OF TYPHUS IN THE NORTH-WEST PROVINCES OF INDIA.
ED. MED. JL., VOL. 6, p. 916, JULY 1860 TO JUNE 1861.

(325) WALKER, W. T.
SCRUB TYPHUS VACCINE.

(326) WEBSTER, W. J.
TYPHUS IN THE SIMLA HILLS: LABORATORY OBSERVATIONS CHIEFLY ON
HUMAN XK STRAINS.
IND. JL. MED. RES., VOL. 37, p. 667, 3RD JANUARY 1940.

(327) WEIGL, R.
INTRA RECTAL INFECTION OF LICE. STUDIES ON RICKETTSIA PROWAZIKI.

(328) WEIL, E.
DIAGNOSTIC VALUE OF AGGLUTINATION REACTIONS WITH VARIOUS
MICRO-ORGANISMS PRESENT IN TYPHUS.
DTSCH. MED. WSNCHR., 16: 343; MARCH 25th, 1920. QUOTED BY FELIX (37).

(329) WEIL, E. AND FELIX, A.
ZUR SEROLOGISCHEN DIAGNOSE DES FLECKFIEBERS.

(330) WEIL, E. AND FELIX, A.
ÜBER DIE BEZIEHUNGEN DER GRÜBER-WIDALSCHEN REAKTION ZUM.
FLECKFIEBER.
(331) WETZEL, U. QUOTED BY ALLEN, A.C AND SPITZ, S. (2)
FLECKFieber UND NIERFENSCHADIGUNG.
ABSTRACTED IN TR. DIS. BULL., 39; 367; 1942.
(332) WAYNON, C.M.
"HARMOGREGARINES" IN MAN, WITH NOTES ON SOME OTHER SUPPOSED PARAZITIDES.
ABSTRACTED IN TR. DIS. BULL., VOL. 20; p. 527; 1923.
(333) WHITNEY, L.E.H. AND BRITTON, C.J.C.
DISORDERS OF THE BLOOD. 5th ED.
(334) WIJEHAHA, E.M. QUOTED BY NICHOLS, L. (219).
(335) WILDER, R.M.
THE PROBLEM OF TRANSMISSION IN TYPHUS FEVER.
JL. INF. DIS., VOL. 9, p. 9; 1911.
(336) WILLIAMSON, J.E.
A POSSIBLE CASE OF TICK TYPHUS.
(337) WILSON, W.J.
ON HETEROLOCOCUS AGGLUTININS MORE PARTICULARLY THOSE PRESENT IN THE BLOOD SERUM OF CEREBRO-SPINAL FEVER AND TYPHUS FEVER CASES.
JL. HYG., VOL. 9, p. 316; 1909.
(338) WILSON, W.J.
THE WEIL-ENLEX REACTION IN TYPHUS FEVER.
JL. HYG., VOL. 19, p. 115, JANUARY 17th 1920.
(339) WILSON, W.J.
FURTHER CONTRIBUTIONS TO THE SEROLOGY OF TYPHUS FEVER.
JL. HYG., VOL. 26, pp. 213-225, JULY 1927.
(340) Wilson, D.A.O.

A case of tropical typhus, complicated by malaria.

(341) Wolff, J.W. Quoting Fraunzyitz, K.

Observations on the well-felix reaction in tsutsugamushi disease.

(342) Wolff, F.K. Quoted by Nicols, L. (219)

(343) Woodhead, L.S.F. and Dutta, U.C.

A note on fevers of the typhus group in Assam.

(344) Wolf, A. and Cowen, D.

Granulomatous encephalomyelitis due to an encephalitozoon (encephalitozoic encephalomyelitis); new protozoan disease of man.

(345) Woolbach, S.B.

Studies on rocky mountain spotted fever.

(346) Woolbach, S.B.

The nickerstck and their relationship to disease.

(347) Woolbach, S.B., Todd, J.L. and Palfrey, F.W.

Etiology and pathology of typhus. 1922.

Published by the league of red cross societies at the Harvard university press, Cambridge Mass.

(348) Yacon, M. bio

Studies of genus proteus; cultural and serological study.
(348) CONTINUED
OF CERTAIN STRAINS OF PHLEBUS GROUP LABORATORY DIAGNOSIS OF
TYPHUS FEVER WILSON-METI-FLIXX REACTION.
IND. JL MED. RES., T. 2, PP. 787-782, JANUARY 1922.

(349) YACHO, N.
A NOTE OF A CIRCUMSCRIBED OUTBREAK OF A TYPHUS-LIKE FEVER
IN BUZARAFGHAN DISTRICT, SOUTH WEST PUNJAB.
IND. MED. CAZ., VOL. 72, P. 565, OCTOBER 1937.

(350) YEOMANS, A., SNYDER, J.C., MURRAY, E.S., ECKE, R.S.,
AND ZARAFOMETIS, C.J.D.
AZOTEMIA IN TYPHUS FEVER.
ARCH. INT. MED., VOL. 25, NO. 5, PP. 741-753, NOVEMBER 1945.

(351) YERSIN, A. AND VASSAL, J.J.
TYPHUS FEVER IN INDO-CHINA.
PHILLIPINE J. SC., VOL. 3, P. 131, 1908.

(352) ZARAFOMETIS, C.J.D.
THE SUSCEPTIBILITY OF RODENTS GERBIUS PYRAMIDUM AND
GERBIUS GERBIUS TO EXPERIMENTAL TSUTSUGAMUSHI INFECTION
(SCRUB TYPHUS).

(353) ZARAFOMETIS, C.J.D., SNYDER, J.C. AND MURRAY, E.S.
IMMUNITY FOLLOWING PARA-AMINOBENZOIC ACID THERAPY IN
EXPERIMENTAL TSUTSUGAMUSHI DISEASE.

(354) ZINSSLER, F.
SUR LA MALADIE DE PELLET LE RESERVOIR INTEREPIDEMIQUE
DU TYPHUS CLASSIQUE.
INST. PASTEUR DE TUNIS, VOL. 23, NO. 2, PP. 149-154, JULY 1934.
(355) ZINNSER, H.

VARIETIES OF TYPHUS VIRUS AND EPIDEMIOLOGY OF AMERICAN FORM OF EUROPEAN TYPHUS FEVER (BRILL’S DISEASE).

AIHER. JI. HYC., VOL. 20, NO. 3. PP. 513-532, 1934.

(356) ZINNSER, H. AND BAYNE JONES, S.

A TEXTBOOK OF BACTERIOLOGY. 8th ED. 1939. P. 653.

(357) ZINNSER, H.; WEI, H. AND FITZPATRICK, F.

AGAR SLANT TISSUE CULTURES OF TYPHUS RICKETTSIAE (BOTH TYPES).

PROC. SOC. EXPER. BIOL. AND MED., VOL. 37, P. 604, 1934.