5. Set up I.V. line if child is dehydrated.
6. Follow guidelines below:

<table>
<thead>
<tr>
<th>Age</th>
<th>Fever, cause found</th>
<th>No neck stiffness</th>
<th>Short convolution</th>
<th>Quick recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>under 2 years</td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Requires LP</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>*refer to hospital</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 - 5 years</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>over 5 years</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of convulsions</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Child well</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

If transfer is likely to be delayed or is refused, and facilities are not available for investigation, it may be justifiable in very sick children to start treatment with penicillin and chloramphenicol, which is the treatment of choice for meningitis, and will also cover severe sepsis and bacterial infection causing febrile convulsions.

ACKNOWLEDGEMENTS

It is with pleasure that we thank the lecturers and consultants who were caring for the children studied, Mrs. E. B. Huntly and Mrs. S. I. Kelly for typing the manuscript and Dr. T. Waterston for the treatment algorithm.

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Plague in Zimbabwe
A Review of the Situation in 1982

by

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The background and the status of plague in Zimbabwe has been fully described by Taylor et al (1981). However the sporadic nature of plague cases in Zimbabwe, as highlighted by the isolated outbreak in the Lupane District of Matabeleland in January 1982, and another case in a different part of the same district in May 1982, suggests the need for a short review of the epidemiology, clinical symptoms and treatment of plague.

EPIDEMIOLOGY OF PLAGUE IN SOUTHERN AFRICA

Plague has been endemic in the wild rodent population of Southern Africa since early this
century (Davis, 1948). It has been suggested that the gerbil Tatera brantsii is the primary reservoir in the wild (Davis, 1953). This may be the case in South Africa but in Zimbabwe the local gerbil Tatera leucogaster and also Praomys coucha are very susceptible to plague usually dying quite quickly (Isaacson, Taylor and Arntzen, unpub. info.) making it unlikely that they can act as reservoir hosts. Rodents such as the rock rat Aethomys chrysophilus or Praomys natalensis which are relatively resistant to plague infection (Isaacson, Taylor and Arntzen, unpub. info.) are more likely to act as sylvatic reservoirs of infection. Praomys coucha is closely related to P. natalensis (Green et al., 1980) but they have quite a different distribution and relative abundance in Zimbabwe (Gordon, 1978). Both P. natalensis and P. coucha are semi-domestic and may act as a link between man and truly sylvatic foci of plague.

The most likely scenario of events in a plague epizootic in Zimbabwe are as follows:-

Plague is present in isolated foci amongst relatively resistant rodent species such as A. chrysophilus or P. natalensis with relatively slow spread from rodent to rodent even when there is a high population of the resistant species.

Under favourable environmental conditions populations of rodent species which are very susceptible to plague (such as P. coucha, T. leucogaster) increase to high levels. If these population increases occur in an area where there is a quiescent plague focus, plague may break out in the susceptible rodent population. Plague kills the susceptible rodents and their infected fleas leave the carcass and seek new hosts thereby spreading the infection rapidly throughout the area of high population.

Dead rodents should be observed – an unusual situation. Cats may be found dead or disappear as they often succumb to plague.

Dogs scavenge and eat dead rodents but although they may develop plague they do not become sick and readily recover.

A week or two after dead rodents are observed human cases may begin to occur. This may be ascribed to rodent fleas, whose rodent hosts have died due to the epizootic, now biting man. Isolated human cases of plague may result from flea bites in isolated pockets of active plague or from accidental infection due to handling infected animals. Because of the sylvatic nature of plague in Zimbabwe it is the rural peasant farmer population who are most at risk from the disease.

**CLINICAL SYMPTOMS**

Human plague is most often contracted from the bite of an infected flea or occasionally by direct contact with the tissues of an infected animal. Both of these forms of contact generally result in the development of classical bubonic plague. The bubo is an enlarged and painful regional lymphadenitis with surrounding cellulitis. The incubation period of bubonic plague is from 2 – 7 days. The initial symptoms usually consist of sudden onset of fever, shaking chills, myalgia, headache and prostration frequently associated with pain in inguinal or axillary areas with or without manifest lymphadenitis. Usually within a few days an excruciatingly painful lymphadenitis (bubo) with overlying and surrounding erythema and boggy tender oedema will develop. The buboes may not be so conspicuous however and may even be missed in mild cases. Because of the non-specific nature of these symptoms, isolated cases may not be recognised and just be recorded as a P.U.O. (pyrexia of unknown origin). The untreated bubonic plague patient may recover spontaneously or progress into septicaemic and secondary pneumonic plague. Septicaemia occurs when the regional lymph nodes no longer succeed in localising the infection which is then disseminated throughout the body, infecting various organs. The resulting septicaemia may be overwhelming and rapidly fatal. Pulmonary infection follows septicaemia in virtually 100% of untreated cases. Functionally transmissible pneumonic infection is estimated to occur in only 5% of such cases since death usually takes place before secondary respiratory spread can be effected from the pulmonary invasion.

The person to person chain of infection which occurs through droplet spread via the respiratory route results in primary pneumonic plague cases. The incubation of such cases is 2 – 3 days and the resulting illness is characterised by a sudden onset of fever; cough (usually with bloody sputum), headache, shaking chills and prostration. This form of the infection is fulminant and, if untreated, results in death in 2 – 5 days. Isolation and contact tracing is important in such cases to prevent further spread of the epidemic.

**LABORATORY CONFIRMATION**

If plague is suspected on clinical evidence treatment must not be delayed whilst waiting for laboratory confirmation. Previous experience (Pugh and Parker, 1975) has shown that examination of bubo material gives the best
immediate results. Aspirate fluid or pus from the bubo or gland, and either examine under Leishman’s stain or fix with alcohol before sending to the Laboratory. Blood culture may be done, or a rising titre of antibodies demonstrated.

TREATMENT

Early treatment of plague is essential and many patients will require intravenous therapy to help combat septic shock. W.H.O. recommends tetracycline as the treatment of choice and high doses (i.e. 3 – 6 g. daily during the first 48 hours) are necessary. Streptomycin is highly effective but the massive destruction of plague organisms may precipitate endotoxic shock. Penicillin is totally ineffective for plague. Sulphonamides are mildly effective and are sometimes used in combination with other antibiotics.

Nursing staff must be protected from the danger of pneumonic plague by masks and chemoprophylaxis. Ordinary barrier nursing procedures are sufficient, but one must not forget that the patient’s clothing may contain infected fleas and should be well fumigated before being stored.

Another important consideration is to remember the risk to laboratory technicians and to send laboratory specimens in sealed double containers clearly marked “Plague”.

COMMUNITY MEASURES

(a) Notification

When a case of suspected plague is seen, the clinician must ensure that measures to identify other cases and protect the community are taken. The best way to do this is to notify the Provincial Medical Officer of Health who will pass the information on to the Secretary for Health and the Blair Research Laboratory. The clinician will probably ask the patient whether anyone else at the place of infection has symptoms, but there is other information which is also vital for the investigators. Hospital and Health Centre staff should ascertain not only the exact address of the patient but also his movements for the two weeks before he became ill because he may not necessarily have contracted the disease at home. It is helpful to know whether an excessive number of rodents have been noticed, alive or dead, as an indication of an active epizootic of plague.

The nearest Health Assistant should be notified by the clinician so that he can begin investigations and explain to the community about the disease as well as being ready to help the plague investigation team when it arrives to consider what control measures need to be taken. So important is it to keep the local community fully informed about the disease and the measures being taken to control it, that it is our practise to take a member of the District Council or Health Committee with us, and also inform teachers in the affected area. Information sheets in both English and Sindebele are distributed as widely as possible amongst the at-risk populations to give them the facts about plague before rumours begin to circulate. The early recognition of plague in humans (buboes) and in rodents (finding dead rats) is valuable.

(b) Chemoprophylaxis

For persons likely to come into direct contact with plague, such as nursing staff, tetracycline in ordinary doses is advised three times a day for at least five days. In the community it is more pragmatic to administer a stat dose of a long-acting sulphonamide. Four tablets of Sulphamethoxypyridazine (Depomide) are given to adults and correspondingly less for children, and they are asked to take one or more tablets for the next three days.

(c) Rodent control

Rodent control may be effected before or during an epidemic. Although the risk of plague could be reduced by preventing rodent populations from reaching high numbers, this is not practical in the widespread forest areas. It can however be helpful in preventing a localised epizootic from spreading to other areas. Rodent poisoning and trapping is best left to the plague team. Rodent control should never be carried out without concomitant flea control otherwise the likelihood of plague transmission to man will be increased.

(d) Flea control

Sometimes this is all that is practical in isolated plague areas, but the protection obtained is probably quite low. Spraying burrows near villages is a hit and miss exercise as they may have many entrances, and it is impossible to cover large areas of forest land. Flea control is most useful in urban plague outbreaks.

PRESENT STATUS OF PLAGUE IN 1982

EPISODE 1

On the 13th January 1982 two sisters aged 8 and 10 were admitted to St. Lukes Hospital, both with fever, patient A with a bubo in the left axilla and patient B with an abscess and oedema
on the right breast. Both initially were given penicillin although patient B was later given streptomycin lanoxin and aminophyllin.

Both patients died the day of admission with septicaemia and evidence of lung congestion. Another sister (patient C) aged 6 years with mild stomach pains had been kept under observation and later given sulphonamides and antibiotics when plague was the suspected cause of her sisters' deaths. Patient C was found to have antibodies to plague at a titre of 1:64 when blood was taken 1 week after the death of her sisters.

Investigations at the home of the index patients and in the general area revealed the following:

(1) The home of the index patients was situated near Tshongokwe mission but in a very isolated location (Grid ref 27° 23'E, 18° 38'S). The patients were said not to have been away from home for several weeks prior to the illness.

(2) The home was constructed in the last year at the end of a line of houses in virgin woodland.

(3) Reports were conflicting but said that the family cat regularly brought rats into the house. The cat was well and seen at the house.

(4) There was no evidence of a recent past or present high rat population in the general area of Tshongokwe. However between St. Lukes and Lupane village were many abandoned burrows of Tatata leucogaster indicating a recent die off in the population. Very few burrows were still occupied possibly reflecting either re-colonisation or a few survivors.

(5) Totjolo clinic, the nearest clinic to Tshongokwe, had recorded several outpatients with suspicious swollen glands. Penicillin was normally prescribed but no records are available of subsequent improvement if any, and very few completed the course. These patients were impossible to trace and it was clear that the personal data recorded by clinics was totally inadequate for this purpose.

Prophylactic long-acting sulphonamides were issued to likely contacts near the home kraal including family and immediate neighbours.

Blood samples were taken from all members of the family of the index cases and also from their nearest neighbours. One dog from a neighbour's kraal also supplied a blood specimen. Sera from these samples were tested for plague antibodies (Swanepoel et al, 1976) and all were negative with the exception of one sister, patient C, as mentioned above.

EVALUATION

It was considered that the index patients represented isolated cases and that the area was not about to suffer an outbreak of plague. Plague had probably reduced the rodent population 3 or 4 months previously and although that should have been the most likely time for human cases to occur, none were recognised. The outbreak was probably related to isolated areas of high rodent population and not widespread in nature. Further isolated human cases were thought possible during the following few months but as the rodent population normally reaches a peak in July/August it is most likely that the plague focus would be reactivated or reach new areas at this time with a concomitantly greater risk of human infection. Further, human plague cases had probably been occurring over the previous few months but not recognised at health centre level.

ACTION

Immediate steps were taken to begin assessment of the affected area using the detection of plague antibodies in dogs and people as the most sensitive indicator. Initial results have shown no antibodies in 394 schoolchildren tested from the immediate vicinity but antibodies in dogs from St. Lukes (9% +ve, n = 22) and Tshongokwe/Totjolo (4.7% +ve, n = 127). Intervening areas were negative, again indicating the patchy nature of the current outbreak. Further sampling and testing is continuing.

EPISODE 2

On the 2nd May 1982 an 8 year-old girl (patient D) was brought to St. Lukes Hospital in Lupane where the experienced mission doctor recognised bubonic plague and instituted treatment immediately with streptomycin and tetracycline. When laboratory specimens proved positive the Blair Research Laboratory and Provincial Medical Officer were notified. The patient made a full recovery. Investigations at the home of patient D revealed the following:

(1) The home was situated near Ndimbimbili dip (Grid Ref. 28° 16'E, 18° 38'S) a sparsely populated area surrounded by woodland.

(2) Rodent burrows, some apparently aban-
should be made to increase community and
arise and may occur in the rural populations of
Lupane, Nkayi and Tsholotsho Districts. Efforts
further isolated cases of plague will probably
burrows. This took a period of 2 weeks.
area was small and a malaria control team was
diverted to apply insecticide to all rodent
affected that further spread of the disease may be
reduced by effective flea control. The affected
small active focus in this case it was considered
acting as a source of infection for rodents in the
human cases were occurring for the reasons
agri~atural
were high in old abandoned fields but not
particularly so in the fields in use, or perimeter woodland. The surrounding
forest had very little evidence of any rodent
population.
rodent
captured at this site were all
Tatera leucogaster. Two of ten captures
died and Y. pestis was subsequently iso­
lated from both animals. Remaining rodent
captures all had high antibody titres to Y.
pestis.
From examination of surrounding country
the epizootic was believed to be restricted
to an area of 4 to 5 square kilometres.
Very few dead rodents had actually been
observed in the area and they had probably
been eaten by wild animals or dogs.
No other people reported any recent illness.

EVALUATION
An isolated epizootic was in progress in the
rodent (Tatera leucogaster) population. Few
human cases were occurring for the reasons
that (a) there were no semi-domestic rodents
(Pr0amys) which could bring the disease into
the home environment, (b) crop harvesting and
therefore contact with the fields was over and
(c) the rodent population was not high enough
to produce a severe epizootic with consequently
many dead rodents and infected fleas. Plague
was considered to be present in the forest,
probably reducing the rodent population there
several months previously, and the forest was
acting as a source of infection for rodents in the
agricultural land. The forest is widespread in
Matabeleland and links Tshongokwe with
Ndimbimbili.

ACTION
Action continued as in Episode 1 but due to the
small active focus in this case it was considered
that further spread of the disease may be
reduced by effective flea control. The affected
area was small and a malaria control team was
diverted to apply insecticide to all rodent
burrows. This took a period of 2 weeks.

CONCLUSION
Further isolated cases of plague will probably
arise and may occur in the rural populations of
Lupane, Nkayi and Tsholotsho Districts. Efforts
should be made to increase community and
health centre awareness of the disease in order
to detect these sporadic cases. It is not con­
considered that an epidemic is imminent or that
there is a risk to travellers.

ACKNOWLEDGEMENTS
We would like to thank the staff of St. Lukes
Hospital, and Mr. A. W. Palmer for their
assistance and the Secretary of Health for
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Sowda Onchocerciasis in Nigeria
by
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College of Medicine
P.O. Box 12003, Lagos.

INTRODUCTION
The disease of onchocerciasis defined by its
cutaneous, ocular and lymphatic features, had
often stimulated interests in the heterogenous
disciplines of the medical profession for over a
century. That the multifactorial spectrum of
this disease still creates
a
challenge to medicine
despite the advancement of science, is a revela­
tion of the vagaries of this parasitic infection.
This parasitic infection was first described
by a surgeon, John O'Neil (1875), in the then
Gold Coast, Africa, and later by a Zoologist,
Rudolph Leukart (1871), quoted by Connor
et al., (1970) and a physician, Manson (1891).
Subsequently Manson (1893), Clapier (1917),
Dry (1924) and Blacklock (1926) described
the disease in other parts of Africa while this
arthropod-borne disease was later described in
America (Robles, 1915, Brumpt, 1919). Recent­
ly, however, Fawdry (1957) had described a
variant of this disease amongst the Arabs in the
Yemen.
Such world-wide description of this rivirine
infection had revealed a high morbidity of the
disease (W.H.O. 1966). While the natural his­
tory of this parasitosis was defined in African