At the time of my writing, the total number of SARS cases in the Hospital Authority (HA) has just dipped for the first time. I hope by the time this issue is published, there will indeed be a sustained decreasing trend.

No doubt, the challenge of SARS to the HA and the public health system in Hong Kong has been unprecedented, even of calamitous proportion. Given the resources that society spends on healthcare, I cannot conceive any decentralized hospital system not already crumbling under such pressure. Thanks to the immense dedication and professionalism of our staff in all the public hospitals, we have been withstanding the almost unmanageable pressure from this crisis so far. The exceptional quality of staff we have is of course the prime and most important success factor. They have paid a big price—their health at risk and that of their family members, their social life severely disrupted, their fear and sorrow, and their extreme tiredness from overwork to help the constant flood of new patients, weeks and months on end. Yet they have persevered with great courage. I am deeply indebted and moved by all of them displaying human behavior of the highest order, given their own personal sacrifice and possible risks. The appreciation and gratitude from the community towards hospital staff come streaming in every day. Our private colleagues have not deserted us, and have indeed been greatly supporting us in all possible ways, for which they also should be praised.

There is little doubt that we are fortunate to have this public hospital system. We have seen how separate hospital systems in some places could not communicate effectively, let alone sharing information with common definition on the same platform and updating knowledge on a daily or even twice daily basis.

Yes there are criticisms on the success rate of treatment, on supplies, on statistics, on manpower deployment, and on just about everything. But remember this is a rapidly spreading disease with a lot of unknowns, requiring hospital treatment with lengths of stay almost five times that of the average patient in acute hospitals, and creating huge emotional challenge to staff. Remember the whole world is scrambling for the same sources of supplies, and for some items we even have to compete with others in Hong Kong. Remember that the emotional challenge applies no less to management staff who always take the blame.

There are indeed criticisms on why healthcare workers keep on getting infected. The usual blame is on supplies. Except in the earlier days, observations in the field usually point otherwise. Infection control teams have discovered environmental factors, and many behavior factors. Basically, we have to re-learn the whole game in caring for patients, in extreme busy wards, and with constant vigilance and enforcement. This is not easy! On top of that, there is the extreme difficulty of balancing the interests of patients and that of staff. Although we have repeated emphasized the need to protect staff first, it is easier said than done in the real world. Imagine a confused elderly SARS patient, coughing and sneezing while refusing to wear a mask, at the point of falling from bed. Do you then reflect on your step 1-7 before approaching him? This is just one example of the moral problems our staff face day in and day out. Very atypical presentations in some patients leading to inadequate alertness among staff also presents another big problem, and unfortunately have been accounting for quite a few clusters of staff morbidity.

In this exceptional battle, we did not have the luxury of preparation, something of paramount importance for all battles. This was Pearl Harbour attack magnified many folds, and in a rapidly escalating pattern. We hardly knew anything about the enemy in the beginning, and we still do not know everything. The situation is therefore novel, and extremely dynamic. There are no known
rules, and therefore the learning has to be extremely fast, and strategies necessarily contingent and dynamic too.

Hence I mentioned the merit of the system. There are daily computerized updates on hospital situations all over the territory, albeit for a disease difficult to define, especially with the atypical presentations. There are daily updated advice to staff and messages to get across, including lessons learnt from individual hospital units to share among all. There is the need to make timely, bold decisions for opening/closure of services to respond to the uneven and sudden upsurge of patients, such as the Amoy Garden incident, with equally rapid corresponding response from other parts of the system to receive new and transfer patients. The volume pressure is so great everywhere that in fact none has real "spare capacity" to help out without trimming services in other areas, obviously with difficulty. Command and control on a large scale are therefore critical.

With hindsight, we have been lucky in our move towards cluster management of hospitals, which had just been accomplished. At least it is far more efficient and effective to involve, agree, and decide among Head Office and seven Cluster chiefs, and through them down the line, than with forty hospital chiefs together. There has also just been time for different hospitals to have worked together for a while, even consolidating services, to face this crisis where internal mobilization and internal cooperation are of utmost importance.

I do not pretend for one moment that we are satisfied. Indeed we are not, and have been constantly frustrated by the worrying situation. However, given the circumstances and limitations, this is probably what one can realistically hope for in any system. On the other hand, great opportunities have been opened up from such crisis. Experts from different hospitals, and indeed among HA and the two Universities, have come together for a common cause. Wisdom and experiences are pooled. Clinicians from one hospital start seeing patients and sharing expert advice to other hospitals. Whole teams have volunteered to help out others in need. Barriers vanished, bonds made.

In another dimension, variations in clinical and management strengths are also highlighted, magnified by the huge system stress. Some are hardly noticeable in the normal humdrum of the day. Many lessons are learned the hard way. These will no doubt point towards improvement directions and strategies in the re-building phase. The foundation and catalyst for necessary change are laid. Opportunities are opened up for long standing problems hitherto undiscovered, to be solved.

Lastly, when we have been previously frustrated by perceiving the lack of appreciation of our work by the public, I believe this is what they are telling us from the bottom of their hearts. Through solid acts of sustained professional dedication, we have won the respect and reputation that we rightly deserve, that in fact had been there all along in the public's mind. Even in the future, we should not be distracted by the noise out there, which masks the unfathomable gratitude that the community truly feels for our noble deeds.

But right now we need to stay united, together with private sector colleagues and the community, in overcoming this big challenge first. We will continue to make use of the system strengths, and a lot of goodwill and selflessness both inside and outside the HA organization, to fight this battle of the century.
Severe Acute Respiratory Syndrome: What Do We Know About This Disease?

Prof. Joseph J. Y. Sung
Department of Medicine & Therapeutic, Prince of Wales Hospital, The Chinese University of Hong Kong

The outbreak of SARS in early March 2003 has shocked the world. In less than 2 months, this emerging disease has affected over 3,000 people from 27 countries. The high infectivity of the virus, the rapid deterioration of pulmonary function, the high death toll of this disease and the predilection for health care workers have created much anxiety of the public as well as the medical profession.

What do we know about this disease? Thanks to the hard work of our virologists, we now know that SARS is caused by a novel Coronavirus. The evidence is compelling and there should be little dispute. We still do not know from which animal(s) did this virus come from. We are not sure about its mode of transmission, although droplet and fomite transmission are likely to be the most important ones. We know that there is a polymorphism within the genome of this virus. We have speculated a lot about its mutations and related this to the different clinical presentations. There is a lot to be explored about this virus. We need the two universities in Hong Kong, indeed the scientific community of the whole world, to work together to study this infection.

What do we know about this disease? From our experience in looking after over 270 patients in the Prince of Wales Hospital and my personal communications with local experts, I believe that SARS can be represented as a tri-phasic disease. In the first week of the illness, most patients presented with fever, myalgia and chills. There is usually no or minimal respiratory symptoms. This is likely to be a viral replicative phase. The virus multiplies in the host cells and being released into the blood stream. Damage in the lung by the virus itself is limited at this stage but cytokines are being produced by the macrophages. The disease progresses relatively slowly in the first week. Some patients may recover spontaneously in this phase of the disease. At least 70% of patients then proceed to the second phase which is an immune hyperactive phase. At this stage of the disease, early response cytokines such as TNFα, IL-1, IL-6 and IL-8 are produced in large quantities. While these cytokines are meant to eradicate the virus, it is also producing a lot of tissue damage in the lung. Patient start to develop respiratory symptoms: shortness of breath (especially in the standing or sitting position), cough with pinkish sputum and chest radiograph shows multiple areas of consolidation involving both lungs. This is a critical period in the illness. If nothing has been done to stop the so-called "cytokine storm", the lung will be permanently damaged and progress to the development of acute respiratory distress syndrome (ARDS). In the third week of disease, the patient will go into the pulmonary destruction phase. Ground-glass appearance of the lung will appear on chest radiograph, arterial blood oxygenation will decline, supplementary oxygen requirement will go up. Eventually, a reasonable oxygenation saturation level cannot be maintained with oxygen therapy, patients will require positive pressure ventilation. For some reasons not entirely clear at this point, patients at this stage of the disease are prone to develop spontaneous pneumothorax or pneumo-mediastinum. If they require assisted positive pressure ventilation, they are also more prone to barotraumas which poses further difficulties to look after these patients. The three phases of the disease are illustrated by the diagram below (Figure 1).

The treatment of SARS is still evolving. Ribavirin is chosen as the anti-viral agent of choice because of its wide-spectrum activities, especially against RNA viruses. To date, the optimal dose of ribavirin to inhibit this Coronavirus is still not known. The major problems of using ribavirin are its teratogenicity and its hemolytic activity. Yet, we have little choices. Corticosteroids are used to combat the cytokines storm. It is prescribed in the second phase of the disease to avoid further lung damage. Corticosteroid should not be used as an antipyretic agent, and obviously not an anti-
viral agent. Methylprednisolone in the form of pulse therapy is effective. Detail protocol can be found in the Hospital Authority Guideline (www.ha.org.hk). Although these recommendations are based on anecdotal experience, controlled randomized study in a life-threatening situation, especially during the crisis, would be difficult. We believe that the key to successful therapy is timing of these medication and prevention of ARDS development. Newer agents, including antiviral agents and immunomodulators, are under investigation. It will take some time before their efficacy can be proven by clinical trials.

**Figure 1.** Severe Acute Respiratory Syndrome (SARS): a tri-phasic disease

**Back to previous page**
Management of Severe Acute Respiratory Syndrome (SARS) in Children

Prof. T. F. Fok
Department of Paediatrics, Prince of Wales Hospital, The Chinese University of Hong Kong

Diagnosis

The predominant and most consistent symptom of SARS is fever, which is present in all the patients so far diagnosed to have SARS. Other symptoms include coryza, and running nose. Chills, rigor, myalgia, and malaise, which are common in adult patients, may also be present in older children and adolescents, but are absent in young children. Some patients, adults and children alike, may present with diarrhea. Young children appear to have milder disease with more speedy resolution, while the disease pattern in older children is more similar to that of the adults.

Since the presentation is non-specific and often indistinguishable from other childhood infections, the diagnosis is often difficult unless there is clear history of contact with an infected patient. The CDC and WHO have promulgated case definition for SARS, and the Hospital Authority has issued criteria for reporting to HA SARS registry. All these definitions and criteria, while being applicable to both adults and children, are based mainly on adult experience. An interim case definition for Paediatric SARS has been promulgated by Dr Leung Chi Wai (PMH) and Dr Li Chi Kong (PWH) on 25 March based on their experience with the first few cases of paediatric SARS. A revised definition was promulgated on 12 April 2003:

Children meeting the definition requiring specific treatment (Antiviral and Steroid) and "Strict" isolation:

1. Fever (rectal temperature >38.5 deg C or oral temperature >38 deg C), AND
2. Chest X-Ray findings of pneumonia or acute respiratory distress syndrome (ARDS), AND
3. Suspected or probable contact with a person under investigation for or diagnosed with SARS, OR exposure to a locality with suspected or documented community transmission of SARS, either through travel or residence, within 10 days of onset of symptoms, AND
4. One or more of the followings: Chills, malaise, myalgia, muscle fatigue, cough, dyspnea, tachypnea, hypoxia, lymphopenia, falling lymphocyte count, or failure to respond to antibiotics covering the usual pathogens of community acquired pneumonia (e.g. a broad spectrum beta-lactum plus a macrolide) after 2 days of therapy in terms of fever and general well being.

Note: Physical findings of prominent crepitations and/or rhonchi on auscultation of chest; Chest X-Ray of lobar consolidation or significant pulmonary effusion; Leukocytosis, neutrophilia or left shift of neutrophils with toxic granulations.

This case definition is extremely useful in guiding Paediatricians in decision making regarding treatment. However, since the early symptoms of Paediatric SARS are no different from other forms of upper or lower respiratory tract infections, the decision on admission, isolation, and treatment of children presenting with fever but without a definite contact history may still be difficult. Sometimes even the contact history may be misleading. In PWH, we have treated two children with symptoms of SARS and clear contact history, who were later diagnosed to have bacterial septicemia. At present, we isolate febrile children at three different levels according to their likelihood of having SARS. The likelihood assessment is based on contact history, symptoms,
presence or absence of pneumonic changes on chest CXR, and investigation findings such as lymphopenia, thrombocytopenia, clotting profile, presence or absence of other pathogens from bacterial and viral cultures of body fluid.

**Investigations**

All suspected cases will be subjected to a battery of investigations: (1) microbiological studies to rule out common pathogens, including blood culture, nasopharyngeal aspirate for immunofluorescence and viral culture, and viral serology; (2) serial complete blood count and differential count which will be repeated daily; (3) serial liver and renal function tests, creatine kinase and lactate dehydrogenase levels; (4) serial clotting profile including prothrombin time, partial thromboplastin time, and D-dimer; and (3) daily chest radiograph. An important point of note is that obtaining nasopharyngeal aspirate is an aerosol generating procedure that may spread the virus. Staff performing the procedure should be adequately protected by wearing mask, gloves, gown, and face shield.

Lymphopenia is quite consistently present in children SARS. There may also be thrombocytopenia, moderately deranged clotting profile, elevated liver enzyme and LDH.

Similar to adults, children with early SARS may have normal chest radiograph but changes of typical airspace consolidation in their CT thorax. However we do not advocate routine CT thorax since this may lead to over-diagnosis as it is possible that other viral infections may also cause lower respiratory tract changes that may be detected by CT thorax. CT thorax will become useful in cases strongly suspected to have SARS (e.g. those with very definite contact history) but with normal chest radiograph.

**Treatment**

The treatment of children with suspected SARS should begin with antibiotics covering both common bacterial (e.g. a third generation cephalosporin such as Cefotaxime) and atypical pneumonia (e.g. Erythromycin or Clarithromycin). Our present practice is to add oral Ribavirin 40-60 mg/kg/day in 3 8-hourly doses if there is definite contact history making SARS very likely. If the symptoms, especially fever and general well being, do not respond to the treatment for 2 to 3 days, steroid will be commenced in form of prednisolone 1-2 mg/kg/day po in 2 divided doses or hydrocortisone 1-2 mg/kg iv 6 hourly. If fever persists or when there is clinical deterioration or progressive CXR change, pulse methylprednisolone 10 mg/kg/dose IV will be given q24h for up to 3 doses, depending on clinical response. Ribavirin will at the same time be changed to 20-60 mg/kg/day IV q8h. Steroid will be continued for a total of two weeks in form of prednisolone 1-2 mg/kg/day or hydrocortisone 1-2 mg/kg/dose IV q6h after methyl-prednisolone. If the child's condition improves, the steroid will be reduced to half the dose and gradually tapered off over a week. However, if CXR is still abnormal by day 21, low dose steroid will be continued for a longer time and slowly tapered off according to clinical and radiological assessment.

The above treatment regime is based on adult experience. The dose and timing of commencement of the two medications are under constant review. In the younger children in whom the infection appears to run a mild course, an obvious question is whether treatment with either medication, either alone or in combination, is of any benefit. This question can only be answered by properly conducted randomized controlled trial.

According to the present policy, all patients with SARS, including children, will be discharged home 21 days after onset provided their condition permits. It is possible that they may continue to shed the virus especially in their excreta after discharge. Instructions should be given to the parents on the proper disposal of excreta in order to prevent further transmission of the disease in the community.