complaints or serological abnormalities (patients have rheumatoid factor, antinuclear antibodies, LE cells, C reactive protein, and increased IgM levels).[3] In-vitro studies have shown that the lymphocyte response to mitogens is depressed in both pregnant women and those taking oral contraceptives,[4] while a combination of oestrogens and progesterone increases the Arthus phenomenon in experimental animals.[5]

Our observation that erythema nodosum spontaneously cleared in the second half of pregnancy suggests that a given concentration of either sex hormones or an optimal ratio between them may be critical to the development of the cutaneous lesions.

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References

1 Elias, P M, *Archives of Dermatology*, 1973, **108**, 716.
2 Hannuksela, M, *Annals of Clinical Research*, 1971, **3**, (Suppl 7), 1.
3 Kay, D R, Bole, G G, and Ledger, W J, *Arthritis and Rheumatism*, 1971, **14**, 239.
4 Hagen, C, and Frøland, A, *Lancet*, 1972, **1**, 1185.
5 Lambotte, R, and Hustin, J, *Comptes Rendus des Seances de la Societé de Biologie et de ses Filiales*, 1974, **168**, 147.

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SHORT REPORTS

Epilepsy and drowning in childhood

Though injuries sustained during epileptic attacks have been well documented, reports on epilepsy-related immersion accidents are few. Thus the Brisbane Drowning Study[1] provided an opportunity to assess the risk in epileptic children. Details of 111 consecutive freshwater accidents in children were included, with an analysis of causes. In addition all 24 cases of saltwater drowning in children in Southern Queensland over the same period were analysed. Data on a further 14 cases occurring in 1970 and 1976 were also available. Necropsy was performed on all drowned victims.

Altogether eight cases (5·4%) occurred as a result of convulsive seizure—three in bathtubs, two in swimming pools, and three in the sea (see table). Out of 76 consecutive childhood drownings, 2 (2·6%) were caused by epileptiform seizures, both in bathtubs.

Case reports

Case 3—A 9-year-old girl who suffered severe uncontrolled grand-mal seizures after a head injury at 18 months and was receiving phenytoin and carbamazepine had attended swimming classes weekly in the summer for seven years. She had a seizure in the water and was rescued from the bottom of the pool by the instructor (immersion time 1–3 minutes). She responded well to mouth-to-mouth resuscitation and spent five days in hospital.

Case 8—A 15-year-old mentally normal girl who had not had a seizure for several years was staying at the home of a sponsor in a student-exchange scheme. She was not taking anticonvulsants. While kneeling over a bath washing her hair under the running tap she had a seizure and slumped into the water. A flannel stopped the water escaping and, she was found drowned after about 20 minutes.

Comment

Epileptic children who swim are four times more likely to drown than normal children, but the absolute risk remains low. When properly supervised there is no evidence that such children are likely to drown or suffer brain damage from anoxia. Although some 400 epileptic children were at risk from drowning each year in Brisbane during the seven-year study, no epilepsy-induced pool or sea deaths occurred. This illustrates the confidence with which such children may be encouraged to swim. No epilepsy-induced immersion occured in a child with controlled epilepsy who had an adequate blood concentration of anticonvulsants. Seizures occurring in the bathtub, however, may be life-threatening.[2]

The role of trigger mechanisms in epilepsy-induced drownings remains unresolved. Hot baths trigger one form of somatosensory-

Details of eight consecutive immersion accidents in epileptic children

Case No	Age	Sex	Pre-accident state	Immersion site	Depth of water	Activity at time of seizure leading to accident	Rescuer	Immersion time	Outcome
1	11 mnth	M	Normal infant. Two febrile convulsions in preceding days	Family bath-tub	23 cm	Child left in bath by mother	Mother	4–6 min	Drowned
2	2 yr 8 mnth	M	Normal child. Feverish and ill on day of accident	Family bath tub	15 cm	Child left in bath by mother	Mother	2–5 min	Severe spastic quadriplegia and amentia. Child institutionalised
3	9 yr 3 mnth	F	Mentally retarded with poorly controlled (1 fit/month) post-traumatic epilepsy Pre-immersion IQ 55	Municipal swimming baths	2 m	Supervised swimming class with other children	Swimming instructor	1–3 min	No apparent clinical intellectual sequelae. Post-immersion IQ 53
4	10 yr 3 mnth	M	Mentally retarded with poorly controlled post-traumatic grand-mal epilepsy	Sea	1 m	Swimming with mother and sibs	Mother	1 min	No apparent neurological or intellectual sequelae
5	12 yr 9 mnth	M	Mentally retarded with tuberous sclerosis and poorly controlled grand-mal epilepsy	Private swimming pool	2 m	Swimming with friends and parents	Bystander	1–3 min	No change from baseline neurological signs
6	13 yr 3 mnth	M	Poorly controlled epileptic. Normal intellect	Sea	1–2 m	Swimming with family	Father	1 min	Complete recovery within 2 days
7	14 yr 4 mnth	M	Gross mental retardation after measles encephalitis. Poorly controlled epileptic	Surf	1 m	Swimming with parents	Father	1 min	No change in intellect
8	15 yr 1 mnth	F	Mentally normal. Well controlled idiopathic grand-mal epilepsy	Bathtub	38 cm	Washing hair, kneeling over bath	Sponsor	10–20 min	Drowned. Did not respond to resuscitation

evoked epilepsy,[3] especially in India, and childhood epilepsy may be produced by undressing before being bathed.[4] Swimming and bathing are known to trigger transient global amnesia. Light shimmering on rippled water may be a factor, particularly in younger patients.[5] Startle effects during horseplay when swimming is another possibility.[5] True water-immersion epilepsy ("bath epilepsy")[4 5] is rare. Infantile febrile convulsions are a threat, especially in view of the common but mistaken parental practice of putting a child who has had a convulsion into a hot bath (cases 1 and 2; see table).

In this study the incidence of epilepsy-induced immersions was 5·4% (epilepsy-induced fatal immersions 3·6%). In Maryland 3·4% of all drownings are due to epileptiform seizures, and in Hawaii 4·6% of sea drowned victims are epileptics. No epilepsy-induced deaths were reported during swimming in a survey of death during sport in South Africa.

Epileptic children should be encouraged to engage in recreation and sport. Competitive swimming, however, may produce somatic stress to the point of exhaustion, which is a trigger for stress epilepsy. Epileptics should never swim or bathe alone (as opposed to showering), and special risks apply to retarded children with epilepsy. With effective supervision, however, epileptic children may swim with confidence.

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[1] Pearn, J H, Nixon, J, and Wilkey, I, *Medical Journal of Australia*, 1976, **2**, 942.
[2] Worster-Drought, C, *Lancet*, 1970, **2**, 876.
[3] Mani, K S, *et al*, *Neurology (Bombay)*, 1974, **20**, 237.
[4] Keipart, J A, *Medical Journal of Australia*, 1972, **2**, 1124.
[5] Mellor, D H, *Developmental Medicine and Child Neurology*, 1970, **12**, 800.

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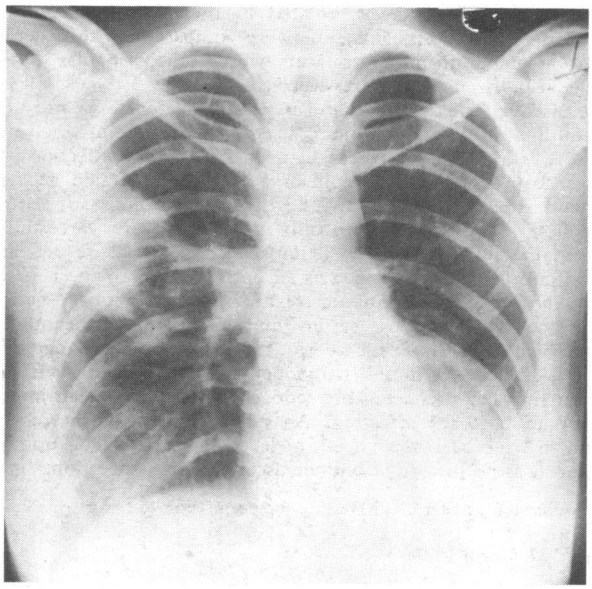
Bacterial septicaemia with multiple organisms complicating influenzal pneumonia

We report a case of fatal bacterial septicaemia secondary to influenza B pneumonia, in which six bacterial species were isolated on two occasions from blood cultures three days after the onset of symptoms. *Staphylococcus aureus* subsequently became the dominant pathogen. Such a phenomenon has not been previously reported.

Case report

A 17-year-old schoolgirl was admitted to hospital in February 1976. She had been healthy until two days previously, when she developed a cough, sore throat, slight hoarseness, coryza, and myalgia. The next day she complained of bilateral pleuritic chest pain and vomited several times. She was admitted on the following afternoon after further deterioration. She had a temperature of 39·5°C, and looked ill and flushed, with a coated tongue, and a sinus tachycardia of 140/minute. Examination of the chest showed bronchial breathing and crepitations in the right midzone and left lower zone. There were no other abnormal physical signs. Chest radiography (figure) showed opacities, suggesting segmental consolidation in the right upper lobe and the left lower lobe, with some patchy changes in the right middle and lower zones. The initial blood count showed a normal haemoglobin concentration of 13·7 g/dl and leucopenia of $3·7 \times 10^9$/l (3700/mm^3); 71% neutrophils, 15% lymphocytes, 11% monocytes, and 3% metamyelocytes. Erythrocyte sedimentation rate (ESR) was 30 mm in the first hour. A blood film showed a left shift of neutrophils. Blood was cultured and she was started on penicillin 2 megaunits twice daily with probenecid.



Anteroposterior view of chest taken on portable *x*-ray machine with patient erect. Opacities suggest segmental consolidation in right upper lobe and left lower lobe, with patchy changes in right middle and lower zones.

At midday on the following day (22 hours after admission) her condition deteriorated suddenly, with circulatory collapse, arterial hypotension, tachycardia, cyanosis, and continuing fever. Chest radiography showed more extensive opacities in all previously affected areas, and the arterial Po_2 was reduced to 4·8 kPa (36 mm Hg). Intermittent positive-pressure ventilation was begun and she was given intravenous methylprednisolone 1·5 g and started on gentamicin and flucloxacillin. The white cell count remained low at $3·6 \times 10^9$/l (3600/mm^3), but two days later had risen to $12·0 \times 10^9$/l (12 000/mm^3) with 87% neutrophils and an ESR of 102 mm in the first hour. Aspiration of tracheal secretions produced scanty but heavily blood-stained sputum. After ventilation and steroid treatment her circulatory state and Po_2 improved slightly, but she remained hypotensive and became oliguric. Over the next few days she developed purpura and conjunctival haemorrhages. Coagulation studies suggested an intravascular coagulopathy, with prolonged prothrombin time, thrombocytopenia, and increased fibrin degradation products. A low-dose heparin regimen was started together with whole-blood and platelet transfusions. After further deterioration over the next few days with falling arterial blood pressure and a rising blood urea concentration she went into ventricular fibrillation, and after rescuscitation developed signs of a brainstem cerebrovascular accident with constricted pupils and bilateral extensor plantar responses. Shortly afterwards she died in asystole, six days after admission. The principal necropsy findings included extensive laryngeal and tracheal ulceration with exudate; bilateral lung consolidation with abscess formation and areas of segmental infarction; petechial brain haemorrhages involving the meninges, cerebral hemispheres, and pons; and acute tubular necrosis. There was also a thrombus on the mitral valve, and two recent splenic infarcts.

Bacteriology and virology—Two sets of blood samples were cultured before starting treatment. Both of these yielded a growth of *Haemophilus influenzae*, *Neisseria catarrhalis*, *Staphylococcus albus*, two species of α-haemolytic streptococci, and Staphylococcus aureus phage type 29/52/52a/80. This organism was subsequently cultured from the sputum and from necropsy material from the mitral valve, trachea, and both lungs. A throat swab was taken three days after admission and inocculated into cell lines MRC 5 and monkey kidney. Influenza B virus was identified in these cells. Viral agglutinin titres on the same day showed nothing abnormal. The influenza B titre was 1/10 but had risen to 1/40 in a specimen of heart blood collected at necropsy.

Comment

The clinical course of events described here is not especially unusual. It shows one of the gravest sequelae of influenza in a young person—the syndrome of viral pneumonia with secondary fulminating bacterial infection and septicaemia—in this case complicated by circulatory collapse, acute tubular necrosis, and disseminated intravascular coagulation. The infecting organism is usually *Staphylococcus aureus* and was, in this case, ultimately the predominant pathogen. The initial yield of multiple organisms from blood cultures is a most unusual feature, however, and we cannot find written reference to it