conversely, have reported it as the presenting feature of the disease after direct pericardial infection.³

In our case the history of arthritis might suggest a preceding meningococcal illness with pericarditis, arthritis, and peritonitis as later complications. The undoubted presence of organisms in the pericardial fluid, however, implies a direct bacterial mechanism.

Corticosteroids have been advocated to aid resolution of the serous effusions of meningococcal infection. Our patient, however, responded satisfactorily to antibiotics and drainage alone. Effective eradication of the organism is obviously the object of treatment and whether steroids offer further benefit seems still open to question.

¹ Murray, E G, *The Urologic and Cutaneous Review*, 1939, **43**, 739.

² Miller, H I, *Israel Journal of Medical Sciences*, 1973, **9**, 1570.

³ Herman, R A, and Rubin, H A, *New England Journal of Medicine*, 1974, **290**, 143.

⁴ Chow, A W, *et al*, *Chest*, 1975, **67**, 611.

⁵ Simon, H, *et al*, *Journal of the American Medical Association*, 1976, **235**, 278.

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The unsupported arm: a cause of falsely raised blood pressure readings

Many technical errors can occur in measuring blood pressure with a sphygmomanometer.^{1,2} We have noticed that many doctors, while measuring blood pressure in a sitting or standing patient, fail to support the arm being measured. Indeed, a survey of 40 doctors in our hospital showed that only five supported the arm. To obtain an accurate blood pressure measurement the cuff must be at heart level.² In the sitting or standing position this means that the upper arm must be extended forward to an angle of 45°. A person holding his arm in this position without support is undergoing isometric exercise, which causes an appreciable increase in blood pressure and heart rate.³⁻⁵ We studied the effects of the arm being unsupported on the blood pressure in normal volunteers.

Subjects, methods, and results

Twenty normal adults (10 men and 10 women) aged 25 to 60 were studied. Their blood pressures were measured by an automated blood pressure device (Arteriosonde 1217-Roche), which uses the ultrasound principle. All blood pressures were measured in the left arm, the upper part of which was extended forward 45° so that the cuff was at heart level. The forearm was parallel to the ground. Three studies, each of which were divided into three stages, were performed. In each study the subject was seated. In the first stage the blood pressure cuff was placed on the left arm, which was then placed on a support with the cuff at heart level, and the patient was allowed to sit quietly for three minutes. During the next four minutes (stage 2) the patient continued to sit quietly but the blood pressure and heart rate were measured every minute. The mean of these four measurements was taken as the control blood pressure. The test period (stage 3) then followed for two minutes: the blood pressure was measured at 30 and 90 seconds and the heart rate every minute. In the first study the left arm was supported at heart level for these two minutes; in study 2 it was unsupported for the two minutes; and in study 3 it was supported but the right arm was raised and

was unsupported. The purpose of the third study was to assess the effect on the blood pressure of isometric contraction in the arm that was not being measured.

The results are shown in the table. The blood pressure and heart rate rose moderately when the arm was unsupported (study 2). In particular, the diastolic pressure measured 90 seconds after the arm was left unsupported increased by 10.6% over the control value.

Comment

Both heart rate and blood pressure showed the greatest increase when the left arm was unsupported. The diastolic pressure rose by up to 10.6%, which may be enough to cause a patient to be considered as hypertensive. Such an increase may also influence the amount of treatment being given.

The change in blood pressure that occurs during isometric exercise is due to a combination of cardioacceleration (due to both reduced vagal and increased alpha-adrenergic stimulation) and increased peripheral resistance (due to increased alpha-adrenergic stimulation).³ The increase in blood pressure after isometric exercise is greater in hypertensive patients than in normotensive ones^{1,5} and is even more exaggerated after treatment with beta-blocking drugs.⁵ Clearly, therefore, great care must be taken in measuring the blood pressure to avoid the isometric exercise that occurs when the arm is left unsupported.

¹ Rose, G A, *et al*, *Lancet*, 1964, **1**, 296.

² Kirkendall, W M, *et al*, *Circulation*, 1967, **39**, 980.

³ Freyschuss, U, *Journal of Applied Physiology*, 1970, **28**, 758.

⁴ Nyberg, G, *Clinical Science and Molecular Medicine*, 1976, **51**, 681s.

⁵ Nyberg, G, *Drugs*, 1976, **11**, suppl No 1, p 185.

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Acute infectious lymphocytosis as a T-cell lymphoproliferative syndrome

Acute infectious lymphocytosis, known in France as *maladie de Carl Smith*¹ because of xenophilia, is an illness (most probably viral) affecting children from 1 to 14 years of age, predominantly young infants. Symptoms are variable but include upper respiratory tract infections, diarrhoea, abdominal pain, and rashes. Lymphadenopathy and splenomegaly are always absent. The ill-defined symptoms contrast with an appreciable peripheral blood lymphocytosis varying from 40 000 to 100 000 × 10⁹/l with a normal haemoglobin and platelet count. We have recently determined the nature of this lymphoproliferation in term of T, B, or null cells.

Case report

A 16-month-old infant, of consanguineous parents, who had been seen at the department of paediatrics for repeated infections, was admitted to hospital with fever and diarrhoea. The result of clinical examination was normal. Initial blood count showed white cells 16.9 × 10⁹/l, with 30%

Mean blood pressure and heart rate (± SE of mean) during 4-minute control period and 2-minute test period in each study

	Blood pressure in control period (mm Hg)		Change in systolic pressure in test period (mm Hg)		Change in diastolic pressure in test period (mm Hg)		Heart rate in control period (beats/min)	Change in heart rate in test period (beats/min)	
	Systolic	Diastolic	30 s	90 s	30 s	90 s		30 s	90 s
Study 1	109.6 ± 2.40	72.6 ± 1.24	0 ± 1.22	+ 0.35 ± 1.2	+ 0.05 ± 0.56	+ 1.30 ± 0.92	73.65 ± 1.63	- 0.35 ± 0.60	+ 0.55 ± 0.68
Study 2	111.8 ± 1.74	73.6 ± 1.78	- 1.25 ± 1.74	+ 2.25* ± 1.74	+ 4.95 ± 1.28	+ 7.80* ± 1.13	73.3 ± 2.19	+ 4.45* ± 0.80	+ 5.25* ± 0.85
Study 3	109.3 ± 3.02	73.10 ± 1.45	+ 3.70* ± 1.09	+ 5.95* ± 1.28	+ 3.95* ± 0.83	+ 5.15* ± 1.39	73.6 ± 1.72	+ 0.9 ± 0.87	+ 2.85* ± 0.73

*P < 0.05.

neutrophils, 1% eosinophils, 64% lymphocytes, and 5% monocytes. Four days later the white cell count was $86 \times 10^9/l$, with 5% neutrophils, 92% small lymphocytes, and 3% monocytes. Bone marrow examination showed an 88% infiltration with small, mature, PAS-negative lymphocytes.

There were no symptoms suggesting whooping cough, and no history of recent vaccination. A chest x-ray film was normal; in particular, there was no suggestion of thymic enlargement. The result of bone marrow cytogenetic analysis and electron microscopy was normal, as was a Paul-Bunnell test. Bacteriological cultures were all sterile, as were all the following antibody titres measured at fortnightly intervals: influenza A, B; parainfluenza I, II, III; adenovirus; ornithosis; psittacosis; respiratory syncytial virus; *Mycoplasma pneumoniae*; Q fever; poliomyelitis I, II, III; herpes; cytomegalovirus; mumps; Coxsackie B1, B2, B3, B4, B5, B6. Immunological investigations showed: IgG 1.120 mg/100 ml, IgA 35 mg/100 ml, IgM 112 mg/100 ml; antibodies to nuclei, smooth muscle, and mitochondria were absent. The lymphocytes were typed by the method of E rosettes for T cells (normal for laboratory $60\% \pm 10\%$) and membrane immunoperoxidase for B cells (normal for laboratory $10\% \pm 5\%$). 78% of the patient's lymphocytes ($56.7 \times 10^9/l$) were T lymphocytes and 5% ($2.9 \times 10^9/l$) were B lymphocytes. Phytohaemagglutinin stimulation showed one-quarter blastic transformation of control. The symptoms resolved rapidly and the lymphocyte count returned to normal in two weeks. The only treatment given was allopurinol to avoid hyperuricaemia. Three months after the infectious episode there were $1.78 \times 10^9/l$ T lymphocytes and $0.22 \times 10^9/l$ B lymphocytes.

Discussion

T lymphocytes play an important part in antiviral immunity and it could be conceived in a genetically predisposed individual that a viral infection might induce a transitory T lymphocytosis. In 1976 lymphocyte typing in a case of acute infectious lymphocytosis was reported² where the authors found equal proportions of T lymphocytes and null lymphocytes. In our case 17% were null lymphocytes and we think that this proportion is insufficient to say there was a "null-cell" proliferation. Indeed, in Sezary's syndrome the median for T cells is 75% and for B cells 3%.³ The same differential exists in our case but is insufficient to postulate the existence of another non-T cell proliferation. If further observations are confirmatory, acute infectious lymphocytosis must be classed among other T immunoproliferative disorders, along with Sezary's syndrome, and or mycosis fungoides, some chronic lymphatic leukaemias, prolymphocytic leukaemias, infectious mononucleosis, and whooping cough.⁴

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¹ Smith, C H, *American Journal of Diseases of Children*, 1941, **62**, 231.

² Dadash-Zadeh, M, Sackett, S H, and Schwartz, A D, *Journal of Pediatrics*, 1976, **88**, 520.

³ Bach, J F, *Immunologie* p 72, Paris Flammarion Medecine Sciences, 1976.

⁴ Griscelli, G, personal communication.

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Intrathoracic tracheal tumour presenting as asthma

Intrathoracic tracheal stenosis may present a difficult diagnostic problem and is often misdiagnosed as asthma. It was recently suggested¹ that flow volume curves should be the mainstay of diagnosis, and subsequently that direct examination is more helpful.²

Case report

A 53-year-old active amateur football referee was first seen as an outpatient at his local hospital in April 1976. He had been forced to give up refereeing two years previously as he developed dyspnoea with a wheeze and tightness across his chest on running 100 yards. In his own words he was "unable to get his breath out." He was a non-smoker. The result of physical examination was completely normal, as was chest radiography, but spirometry showed forced expiratory volume in one second (FEV_1) 2.69 l (predicted 3.82), forced vital capacity (FVC) 6.58 l (4.9), and FEV_1/FVC ratio 40% (78%). Exertional asthma was diagnosed.

He was lost to follow-up but was admitted to his local hospital in January 1977, with a three-month history of progressive dyspnoea with wheeze and a two-week history of cough with sticky white sputum. He was febrile, dyspnoeic at rest, though not cyanosed, and had generalised high-pitched expiratory rhonchi on auscultation. Pneumococcus was grown from his sputum, and chest radiography showed right basal pneumonia. He responded poorly to treatment and needed ventilation for eight days via a cuffed endotracheal tube. He was finally discharged after three months, when chest radiographs were clear, but spirometry showed FEV_1 0.50 l, FVC 1.80 l, and FEV_1/FVC ratio 28%, not improved by bronchodilators.

His convalescence in Devon was interrupted by a recurrence of his wheezy dyspnoea after a heavy cold, and in May 1977 he was admitted to this hospital. On admission he was dyspnoeic at rest with pronounced expiratory stridor. He was afebrile, but had a tachycardia of 120 with pulsus paradoxus. There was a generalised expiratory wheeze, loudest over the trachea. He was otherwise normal.

Tracheal stenosis, probably secondary to controlled ventilation via a cuffed endotracheal tube, was provisionally diagnosed. The patient was treated with antibiotics, steroids, and salbutamol and was referred to the otolaryngologist. The cause of the obstruction could not be seen on mirror examination. Tracheal tomograms were considered, but his condition was thought to necessitate urgent tracheostomy. At tracheostomy an annular tumour was seen just above the carina and distal to the tracheostomy tube. An extended tube was passed through the tumour. The histology of the tumour was adenocystic carcinoma (cylindroma) of the trachea.

At thoracotomy five days later the tumour was found to extend down past the carina into both upper main bronchi. Lower tracheal and carinal resection was performed with reconstruction by anastomosis of the right main bronchus to the trachea and the left main bronchus to the side of the right main bronchus. Postoperatively the patient made a good recovery.

Comment

Intrathoracic localised airways obstruction may masquerade as asthma^{3,4} or chronic obstructive airways disease. The interval between presentation with symptoms and correct diagnosis varies between one month and six years.⁴ Yet it is an important diagnosis to consider, as stenosis secondary to trauma from cuffed endotracheal tubes is likely to become more common, and locally invasive tumours may be successfully treated with surgery or radiotherapy if diagnosed early.

Flow-volume loops⁴ and the comparison of maximum inspiratory with maximum expiratory flow rates⁵ have been suggested as diagnostic tools. The case for direct examination has been succinctly stated.² A history of dyspnoea with wheeze or stridor on moderate exertion, the absence of symptoms at rest, and an abnormally low FEV_1 and FEV_1/FVC ratio should arouse suspicion. The definitive investigations then are tracheal tomography and endoscopy.

¹ *British Medical Journal*, 1977, **1**, 669.

² Grant, I W B, *British Medical Journal*, 1977, **2**, 1157.

³ Baydur, A, and Gottlieb, L S, *Journal of the American Medical Association*, 1975, **234**, 829.

⁴ Harrison, B D W, *Quarterly Journal of Medicine*, 1976, **45**, 625.

⁵ Pride, N B, *British Journal of Diseases of the Chest*, 1971, **65**, 135.

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