VLA-4 or LPM-2 participates, for example, in the inflammatory response in asthma but does not contribute to a minor extent at all to the homing of lymphocytes to Peyer’s patches.2 ICAM-3, a member of the immunoglobulin superfamily and constitutively expressed on lymphocytes and in intestinal Langerhans cells, should be added to the list of counter-receptors of LFA-1 since it has been cloned already in 1992.4 CD21, TAPA-1, Leu-13, and CD19 form a very large number of superfamily B lymphocytes that upon crosslinking transmits signals into the cell but has not been identified to function as a ligand for B4 integrins in adhesive inter-actions.5 A recently described novel LAD syndrome termed LAD type 2 deserves being mentioned.6 This complex syndrome is characterised by recurrent bacterial infections with high neutrophil counts, mental retardation, short stature, and the Bombay blood phenotype. The analysis at the molecular level revealed the lack of expression of Sialyl-Lewis X (sialylated form of CD15), the carbohydrate ligand of the leucocyte adhesion molecules E-selectin and P-selectin, on haemopoetic cells. Due to this defect neutrophils are no longer capable of adhering to endothelial cells using the E- and P-selectin pathway which initiates the transmigration of immunocompetent cells through the blood vessel. The underlying mechanism leading to the deficiency of Sialyl-Lewis X probably is a genetic defect in the fucosylation of glycoproteins and carbohydrates. The disturbance of the Bombay phenotype would also explain the Bombay blood phenotype and may lead to the understanding of the other components of the syndrome as, for example, the mental retardation.

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Dr El Habbal and Professor Strobel comment: We thank Dr Wagner for his interesting comments on our review on leucocyte adhesion deficiency (LAD) which focused on clinical presentation and management.1 We do not intend to discuss extensively the interconnection of homology of adhesion molecules and, for example, did not include the cadherins2 as a fourth family of adhesion molecules. The increase of ‘consensus’ data published, and single reports, however interesting in their own right, were not included and ICAM-3 and the recently described novel ‘LAD type 2’ were consequently not discussed. We did not associate LPM-1 with CD28 as Dr Wagner states. It is B7 (CD80) (and not β7 as erroneously printed in our table 1) which associates with CD28. We apologise for this misprint. His comments on LPM-1 and LPM-2 are correct, but they apply to mouse integrins only.

VLA-4 is not recognised to be abnormal in LAD. The VLA family of integrins are expressed as heterodimers, composed of a variable α (CD49a, b, c, d, e, f) and a constant β chain (CD29). Human VLA-4 (CD49d) is known to be ‘promiscuous’ and known to associate with either β1 and β2 integrin subunits. In the mouse CD49d can pair with β7 (as described before) as well as with integrins αL/β2, αM/β2, and αV/β2 integrins, respectively. In humans, LPM-1 shares homologies with β1 and β6 integrins, as stated.3 Dr Wagner’s remarks on P-selectins and E-selectins, as well as his comments on a signal transduction complex (CD21, TAPA-1, Leu-13, CD19) are interesting but not relevant to the topic of our review.

LAD is a key example where arsute clinical observations (combined with basic research) have led to an exponential increase in our understanding of the role of cellular adhesion pathways in innate and adaptive immunity (‘upora non agunt nisi fixata’; Paul Ehrlich).

MIGRATION OF FINE BORE SILASTIC CATHETER TO PULMONARY ARTERY

EDITOR,—We wish to report a serious complication that arose during the insertion of a line, which was to be used to administer intra-venous antibiotics to a 12 year old boy with cystic fibrosis. A fine bore Silastic neonatal catheter (Epicutaneous-Cava-Catheter model No. 2184, Vygon) was used. This model has a detachable Silastic indwelling catheter which is inserted through a supplied 19 gauge needle. The proximal end of the catheter contains a short metal rod that is anchored within the distal end of the external supply line to pre-vent migration once sited. After veneupuncture of the left basilic vein the catheter, unattached to the supply line, was threaded through the needle. The catheter was almost fully advanced so that the distal end might lie in a satisfactory position within the great veins. The needle was then removed from the vein and disengaged from the catheter by threading it with LPM-2 catheter. At this point the catheter was in normal vision and when the needle was fully removed, the proximal end was still not visible. Chest radiography revealed the metal proximal tip of the catheter to be lying in the right pulmonary artery. The catheter itself was radiolucent. We presume that dynamic venous flow and negative intrathoracic pressure caused aspiration of the whole catheter into the circulation. Subsequently, the patient underwent general anaesthetic and cardiac catheterisation.

The catheter was successfully removed intact using a snare wire during a difficult and lengthy procedure. We have used this type of line successfully for a number of years. The manufacturers inform us that this complication has occurred once before worldwide. They also stated that adherence to the instructions included with the line, updated in April 1992, should prevent this complication. The instructions state that, after advancing the catheter through the needle to the desired position, ‘the catheter should then be fixed in its final position by applying slight pressure beneath the needle tip and the needle is then withdrawn’. However, there is no warning in the instruction leaflet of the potential complication reported here, although we gather that this will be highlighted in future.

The use of short and long indwelling venous catheters is known to carry a small risk of embolism to the heart and great vessels and has in the past led to both serious morbidity and mortality due to thrombus formation, infection, and perforation.1 For these reasons, attempted removal of embolised catheters or fragments is recommended. This has been achieved by bronchoscopy2 or by cardiac catheterisation.3,4

Embolism has been more commonly reported with use of the ‘catheter in needle’ cannula where during insertion the needle shears off the distal part of the catheter.1,4 This complication has led to widespread use of ‘needle in catheter’ forms of cannulas, although these are not free from risk of fracture and embolism.5 Embolism due to disconnection of the catheter from the supply line has also been described6 and most catheters now in use have undetachable external hubs to prevent this problem. The catheter used in this report might have avoided the above safety features. For such lines to be used safely, it is necessary to ensure that throughout insertion the line is at all times both visible and held externally.

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Simultaneous pulmonary infection with respiratory syncytial virus and human cytomegalovirus

EDITORS.—Respiratory syncytial virus (RSV) is the major cause of acute lower respiratory infection in infants and young children. The presentation and subsequent course of RSV bronchiolitis may be atypical in the presence of a simultaneous infection with other viral agents.1 2 During the winter 1991–2, we studied children hospitalised for respiratory disease in a paediatric unit in Marseille. All patients were tested for viral infections and an information chart was made to determine the prevalence of multiple viral isolates and to assess the impact of dual infections on the severity of clinical disease.

Between December 1991 and February 1992, 405 children were hospitalised for respiratory disease. In all cases, nasopharyngeal wash specimens were taken on admission to the paediatric unit and simultaneously submitted to human cytomegalovirus isolation and respiratory virus fluorescent antibody staining. For human cytomegalovirus isolation, specimens were inoculated on human embryonic lung fibroblasts and a monoclonal antibody directed against the immediate early antigen (E13, Biosoft, Clonatec, France) was added 48 hours later to detect viral antigen expression. Simultaneously, indirect immunofluorescence assay was performed directly on nasopharyngeal secretions, using monoclonal specific antibodies against several viruses: RSV, influenza A virus, influenza B virus, parainfluenza virus type 1, 2, and 3 (Monolhu kit, Pasteur, France), and adenovirus (Biosoft, Clonatec, France).

The following information was obtained for each patient: age, sex, history and clinical symptoms, other infections, duration of hospitalisation, socioeconomic status, and ethnic group.

From the 405 children hospitalised for bronchiolitis or respiratory disease, 195 (48%) presented viral infection: 165 were positive for RSV, 30 were positive for human cytomegalovirus, and 14 had a simultaneous infection with RSV and human cytomegalovirus.

Given the frequency of RSV-human cytomegalovirus coinfection in our series (10%), we studied these 20 children. The sex ratio was 1:1 and they were aged 1 month to 8 years. Fourteen of them were less than 4 months old, hence they may have had congenital or perinatal human cytomegalovirus infection. However, six were from 6 months to 8 years old. One child had oral candidosis and one was simultaneously infected with Haemophilus influenzae and rotavirus, one had a history of pneumonitis, but all were without underlying disease. Most were from low socioeconomic groups. Three were of Spanish extraction, seven were North Africans, and two were black children. No specific clinical situation was correlated with the coinfection: five had fever over 39°C, four had severe bronchitis or pneumonitis requiring corticotherapy. But the severity of the children’s illness was demonstrated by the duration of hospitalisation: the average was 16 days compared with 3–2 days in RSV isolated infection.

We especially noted the frequency of RSV-human cytomegalovirus simultaneous infections in the children hospitalised for lower respiratory tract infections. The persistence of human cytomegalovirus in bronchiolitis remains unclear3 4 and further clinical and biological investigations should be undertaken.

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Carers as children

EDITORS.—Aldridge and Becker in their article comment that there is uncertainty as to how many child carers there are.1 We have recently completed two surveys into the health and social care needs of assessment of people with multiple sclerosis in Bradford and Huddersfield. We surveyed over 500 people who agreed to volunteer. Of those agreed to be involved, 74% were aged over 20% between the districts indicated that their children helped with either domestic or personal care.

A further random sample of 192 were invited to complete by face interview. Approximately 40% of those with children under 16 years felt that their children helped more than they normally would with personal care. However, from the parents’ point of view it did not all seem to be bad. Approximately 40% felt that the multiple sclerosis had had little or no effect on their