VLA-4 or LPAM-2 participates, for example, in the inflammatory response in asthma but does only contribute to a minor extent if 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 intradermal 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 complex on the surface of B lymphocytes that upon crosslinking transmits signals into the cell but has not been identified to function as a ligand for $\beta 4$ integrins in adhesive interactions.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 haemopoietic 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 general defect in the fucosylation of glycoproteins and carbohydrates. The disturbance of the fucose metabolism 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 did not intend to discuss extensively the interspecies homology of adhesion molecules and, for example, did not include the cadherins² as a fourth family of adhesion molecules.

Our review was based on '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 LPAM-1 with CD28 as Dr Wagner states. It is B7 (CD80) (and not $\beta 7$ as erroneously printed in our table 1) which associates with CD28. We apologise for this misprint. His comments on LPAM-1 and LPAM-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 $\beta 1$ and $\beta 7$ integrin subunits. In the mouse CD49d can pair with $\beta 7$ or $\beta 1$ chains to generate LPAM-1 and LPAM-2. In humans, LPAM-1 shares homologies with $\beta 1$ and $\beta 6$ 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 astute 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 ('*copora non agunt nisi fixata*', Paul Ehrlich).

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Migration of fine bore Silastic catheter to pulmonary artery

EDITOR,—We wish to report a serious complication that arose during insertion of a long line, which was to be used to administer intravenous antibiotics to a 12 year old boy with cystic fibrosis. A fine bore Silastic neonatal catheter (Epicutaneo-Cava-Catheter model No 2184, Vygon) was used. This model has a detachable Silastic indwelling catheter that 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 prevent migration once sited. After venepuncture 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 over the proximal end. At this point the catheter was lost to 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 thoracotomy^{2,3} or by cardiac catheterisation.^{4,5}

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.^{1,6} 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.⁷ Embolism due to disconnection of the catheter from the supply line has also been described^{1,7} and most catheters now in use have undetachable external hubs to prevent this problem. The catheter used in this reported case has neither of 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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Prophylaxis or modification of varicella by oral acyclovir after household exposure

EDITOR,—In his commentary on our paper Dr Conway raised several concerns about the use of oral acyclovir in varicella infection.¹ We do believe that varicella should be