flat nasal bridge with a small snub nose, but the overall pattern of the face does not seem to fit Robinow syndrome as I have seen it in a total of seven cases.

First, the frontal bossing is not impressive and actually looks more like a metopic ridge. Infants with Robinow syndrome usually have pronounced frontal bossing and more obvious macrocephaly than case 1 of Sabry et al. Her head also may seem large as a result of the obvious dystrophic condition she is in.

Second, the lower face is too fine and the chin too pointed for Robinow syndrome. The mouth does not look like the typical thin lipped “carp mouth”. The overall facial structure is too finely sculptured. The face in Robinow syndrome tends to be rather coarse, Greig hypertelorism-like, in contrast, with a rounded lower half. The coarseness can be so pronounced that sometimes metabolic investigations are initiated because a mucopolysaccharidosis is suspected (own observation).

Third, eyelid hypoplasia giving the impression of exophthalmos seems to be quite a common feature, but I do not see it in this patient.

Fourth, the mesomelia in case 1 is certainly not impressive. It is a highly variable feature and actually not of much use in the diagnosis (see, for instance, Bain et al.), but if present it is an argument for the diagnosis, so some measurements of bone length would have been helpful here.

Finally, stating that the labia minora and clitoris were “slightly hypoplastic” seems too vague. What is “slightly”? A photograph would have been helpful.

The photograph of subject 2 in fig 7 poses some difficulties. Though it is true that the facial abnormalities tend to become somewhat less obvious with age, some anomalies remain quite obvious: the snub nose, the hypertelorism, and the thick alveolar ridges. The face also remains rather square and coarse. Subject 2 has a large nose compared to some of my patients of the same age and his hypertelorism is rather modest. His face seems too fine, much like his sister’s. His alveolar ridges can, of course, not be judged from the picture. In my opinion, a diagnosis of Robinow syndrome is not certain in this case either.

As far as case 2 is concerned, her photograph (fig 8) is more convincing. Particularly when comparing the lower half of her face with that of case 1, it will be seen that there is a clear difference between the two. In my opinion the face of case 2 is definitely more “Greig-like”. Though there is no mesomelia in this patient, this feature can be as variable, as stated. I feel that in this case the diagnosis of Robinow syndrome is probably correct. This patient is not related to the other patients and, considering this, I think it is possible that the authors have in fact encountered, in case 1 and subject 2, a new recessive malformation syndrome with some resemblance to Robinow syndrome.

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This letter was shown to Dr Sabry et al, who reply as follows.

We read the comments of Dr van Steensel regarding our report of unusual traits associated with Robinow syndrome. In family 1 of our report, we described a female patient with many of the constant traits of Robinow syndrome, who showed other unusual traits in addition. We also observed variable expression of some of the traits of Robinow syndrome in healthy sibs/cousins of the proband in this consanguineous family. Like many syndromes for which no molecular/biochemical/cytogenetic markers have been identified, the Robinow syndrome remains solely dependent on the clinical phenotype of the patients. Naturally, this gives wide scope for different subjective views to argue for or against a given diagnosis. This is particularly true for Robinow syndrome, with its wide spectrum of inter/intrafamilial phenotypic heterogeneity that would be expected to reflect a corresponding degree of molecular variability. Of course, the profile in the proband of family 1 does not show a straightforward Robinow phenotype, or it would have been of little interest to the genetics community. Although we bear in mind the possibility of a new Robinow-like malformation syndrome in family 1 of the report, we are reluctant to designate it a new syndrome until all available possibilities are exhausted. Incidentally, we have recently received a letter from Dr H G Brunner from the Department of Genetics, University Hospital, Nijmegen, expressing interest in our Robinow syndrome cases and requesting our collaboration in their ongoing molecular study to map and clone the gene(s) responsible, which we are now considering.

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BOOK REVIEW

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This book reviews the genetic changes observed in solid tumours with particular emphasis on the practical issues of diagnosis, prognosis, and monitoring therapy. The book provides comprehensive coverage of the impact of the new genetic technology in fur-